Dissertation on

"A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS"

Dissertation Submitted to
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,
CHENNAI.

With Partial Fulfillment of the Regulations
for the Degree of

MASTER OF SURGERY

BRANCH-1 (GENERAL SURGERY) AT

MADRAS MEDICAL COLLEGE, CHENNAI.

MADRAS MEDICAL COLLEGE,
CHENNAI.

APRIL 2017

CERTIFICATE

This is to certify that the dissertation titled "**A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS"** is the bonafide work done by **Dr. J.MOHAMMED FAROOQ**, during his M.S. General Surgery course 2014-17, under my guidance and supervision in partial fulfillment of the rules and regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University, Chennai for M.S. (Branch-I) general surgery Examination, April 2017.

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DECLARATION

I, **Dr. J.MOHAMMED FAROOQ,** certainly declare that this dissertation titled "**A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice and are acknowledged. I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, either in India or abroad. This is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery degree Branch 1 (General Surgery).

Dr. J.MOHAMMED FAROOQ

Date:

Place:

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CERTIFICATE OF APPROVAL

To
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Dear Dr.J.Mohammed Farooq,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS" - NO.11022016.**

The following members of Ethics Committee were present in the meeting hold on **02.02.2016** conducted at Madras Medical College, Chennai 3

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We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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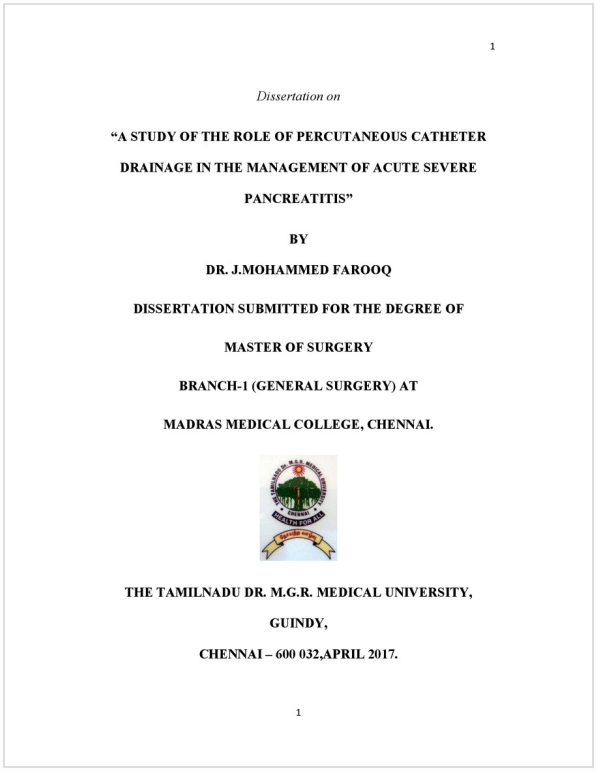

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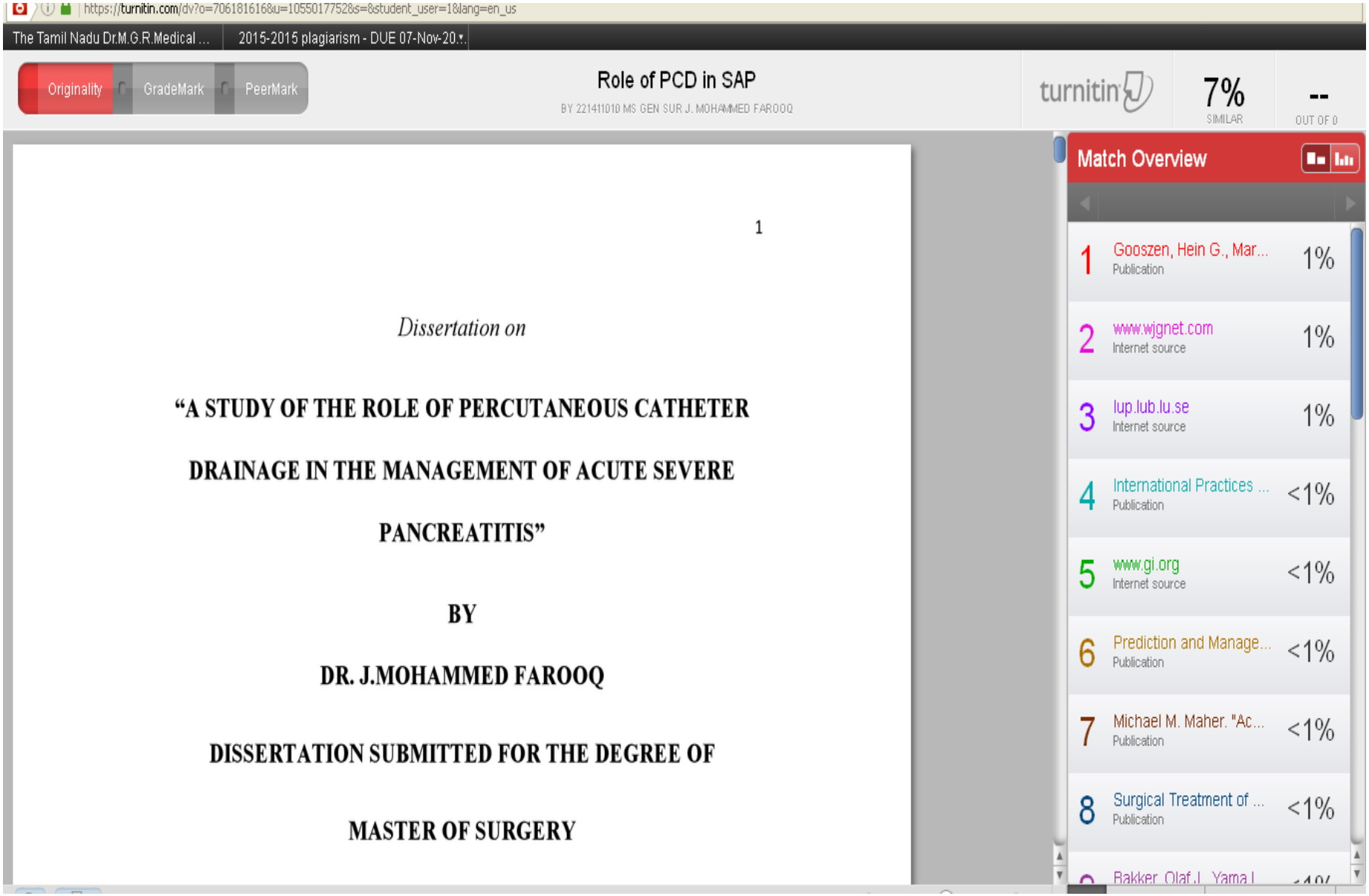
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CONTENTS

INTRODUCTION

"Acute pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera. The suddenness of its onset, the illimitable agony which accompanies it, and the mortality attendant upon it, all render it the most formidable of catastrophes"

Lord Moynihan, 1925

Acute pancreatitis is an inflammatory process of the pancreas, with variable involvement of peri-pancreatic tissues and remote organ systems. In majority of the cases, the disease process is mild, with interstitial oedema and therefore leads to recovery within a few days or weeks. On the other hand, severe pancreatitis, which is characterized by local or systemic complications, is very demanding and associated with severe morbidity and even death, in nearly 15-20% of the cases.

The treatment of acute pancreatitis is not disease-specific, targeting underlying pathophysiology. Initial life-saving measures include fluid resuscitation and supportive therapy to various organ systems which mitigates the concomitant course of the disease process thereby decreasing the severity of acute pancreatitis.

There has been a paradigm shift in the treatment protocols of acute pancreatitis over the past two decades. The management has shifted from an early aggressive surgical treatment to a more conservative strategy. Interventional treatment is seldom needed in majority of the patients with acute pancreatitis. However, interventional treatment in the form of percutaneous catheter drainage, open or minimally invasive endoscopic necrosectomy is indispensable in certain conditions of severe acute pancreatitis.

In this study, we assess the role of image guided percutaneous catheter drainage of peri-pancreatic fluid and necrotic collections in the management protocols of severe acute pancreatitis.

This study was conducted at the Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital.

AIMS AND OBJECTIVES

AIM

To assess the role of percutaneous catheter drainage in the management of severe acute pancreatitis.

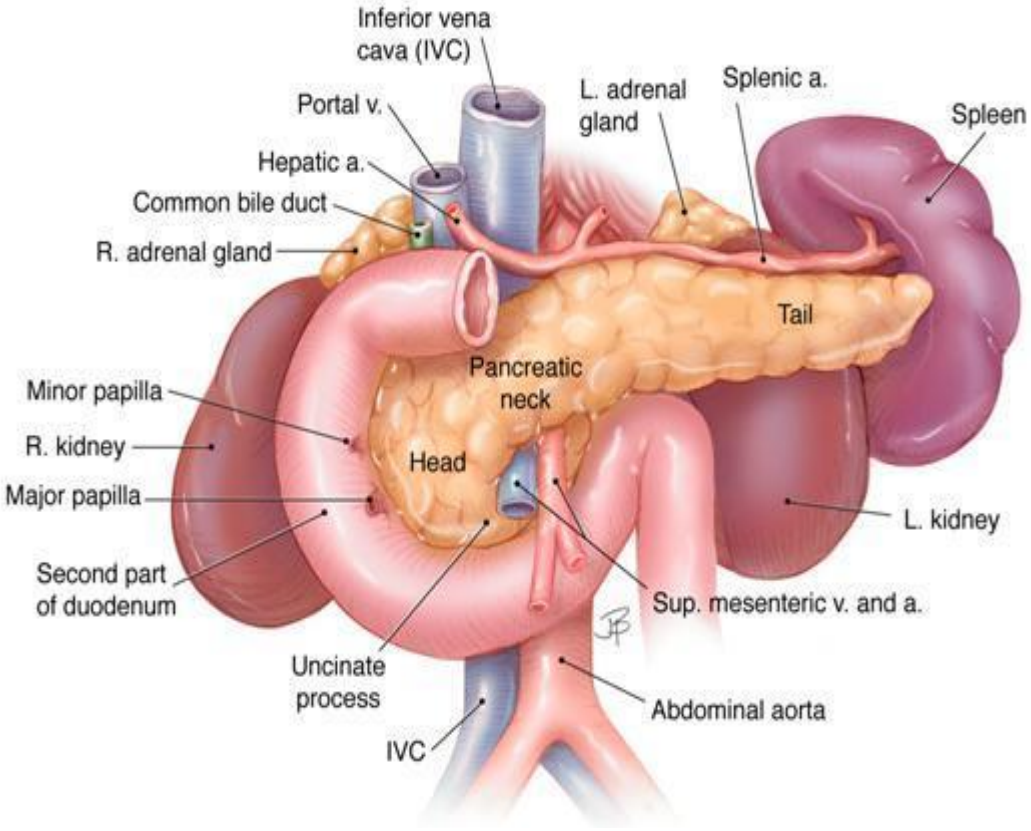
OBJECTIVES

- To compare and analyze the outcome of patients with severe acute pancreatitis managed by conservative treatment, percutaneous catheter drainage and surgical necrosectomy.
- To compare the role of each management strategy in sterile and infected necrotic pancreatitis
- To study the pros and cons of different treatment groups with particular emphasis on the benefits of percutaneous catheter drainage.

REVIEW OF LITERATURE

Herophilus of Chalkaidon first described pancreas as early as 300B.C. In 100 A.D., the organ *pancreas* (Greek: *pan*, all; *kreas*, flesh) was named by Rufus of Ephesus. Alberti et al. in 1578 recorded a series of patients dying of inflammatory conditions or cancer of the pancreas. In 1889, Fitz reported the first classification system for acute pancreatitis. In 1901, Opie related gallstones as a causative factor to acute pancreatitis and in 1917, alcohol was identified as an important causative factor. Hans Chiari postulated that intra-pancreatic zymogen activation causes pancreatic auto-digestion and is an important factor in the development of acute pancreatitis. The link between elevated amylase levels and acute pancreatitis was realized in the early part of the 20^{th} century. As for as the radiological history is concerned, the pancreas was essentially a concealed organ which can only be visualized indirectly by upper GI barium swallows and barium meal examination. Abdominal ultrasonography was the first investigative modality that allowed direct pancreatic imaging. However, imaging of the pancreas developed exponentially with the advent of computerized axial tomography of the abdomen. The indications for surgical management of severe acute pancreatitis has changed over the last few decades. In earlier reports, total pancreatectomy was performed which caused unacceptably increased

mortality. In the late 19th century, open puncture and drainage became the first surgical management for a cyst of the pancreas cyst which was probably a pseudopancreaticcyst. The current concept states that, surgery, offering removal of the non-viable and necrotic tissue benefits those patients. Moreover, surgery is also indicated when the conservative management fails to improve the MODS.



ETIOLOGY OF ACUTE PANCREATITIS

Acute pancreatitis has been attributed to a wide range of causative factors such as

Metabolic:-

Alcohol

Hypercalcemia

Hyperlipidemia

Drugs like azathioprine, thiazides, aminosalicylic acid

Genetic

Mechanical:-

Cholelithiasis

Postoperative

Pancreatic divisum

ERCP

Post traumatic

Pancreatic tumour

Ascaris lumbricoides

Pancreatic ductal bleeding

Duodenal obstruction

Vascular:-

Postoperative (e.g. cardiopulmonary bypass)

Atheroembolism

Polyarteritis nodosa

Infection:-

Mumps

Coxsackie

Cytomegalovirus

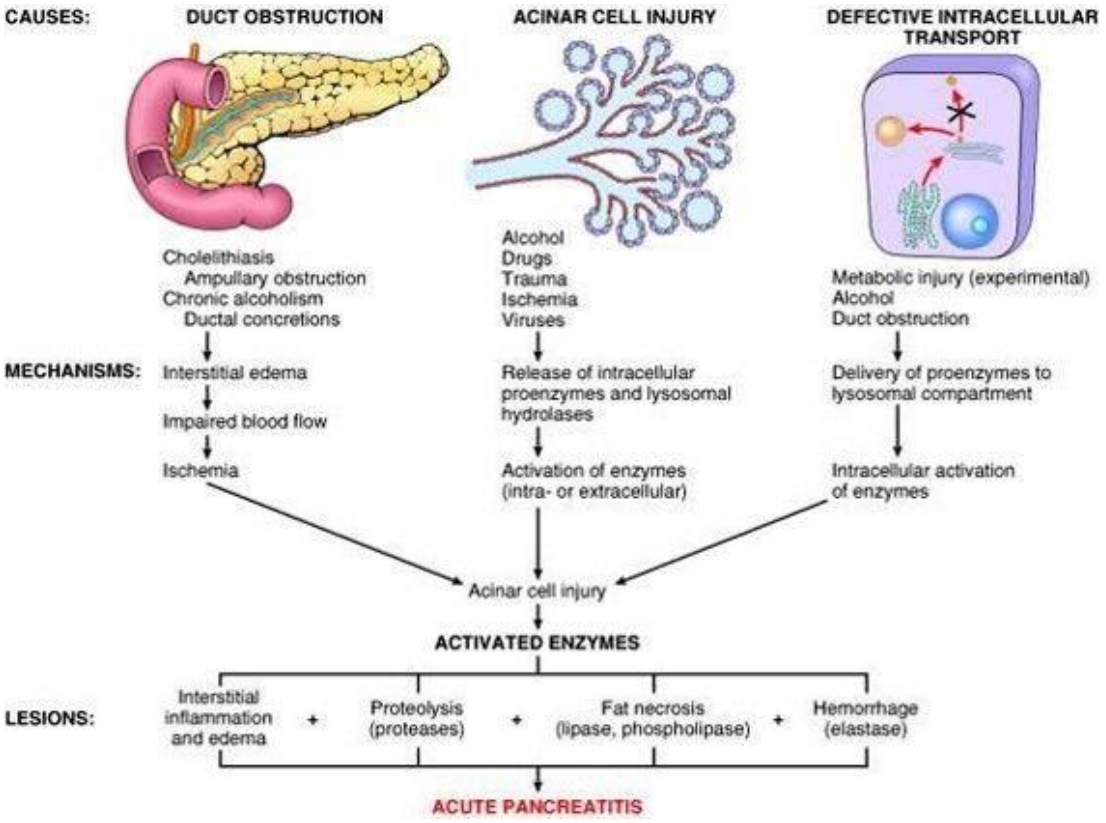
Cryptococcus

PATHOLOGY OF ACUTE INFLAMMATION OF PANCREAS

INITIAL EVENTS

The main role of acinar cells of the pancreas is the formation and release of digestive enzymes in an inactive state. The zymogens released into the second part of duodenum include pro-carboxy peptidases A and B, pro-elastase, trypsinogen, chymo-trypsinogen, and pro-phospholipase A2. Inactive precursors, produced in the RER are packed within the golgi apparatus to be secreted into the small intestine. The granules are then released into the lumen of the acini following its stimulation which are collected by the ducts of the pancreas to be secreted into the second part of the duodenum where its activation happens. The pro-enzymes are activated mainly by trypsin. The 2 important iso-enzymes of trypsinogen

include trypsinogen 1 and trypsinogen 2. In normal individuals, trypsinogen 1 is four times more than trypsinogen 2 in the secretions of pancreas. The proteolytic breakdown of TAP (trypsinogen-activation peptide) results in the activation of trypsinogen. Due to the remarkable capacity of proteo and lipolysis, trypsinogen can auto-digest the whole pancreatic tissue. The safeguarding strategies of the pancreas include intra-cellular transport, production of proteolytic proteins as inactive precursors, along with enzymes that prevent the intracellular activation of zymogens. The pathophysiology of acute pancreatitis is not fully understood. The first stage includes mechanisms (extrapancreatic in origin) like gall-stone blockage or alcohol consumption that initiate the disease process. The major step causing cellular damage is an enzyme from lysosome, namely cathepsin-B which activates trypsinogen. Such an irreversible process causes acinar cell dysfunction. The activated enzymes cause necrosis when it overflows into the interstitial spaces of the pancreas, the retro-peritoneal and peritoneal cavity and into the blood-stream by proteolytic and lipolytic activity.



SECONDARY EVENTS

The pathophysiology of acute pancreatitis is only partially understood by the premature activation of pancreatic enzymes. The next step in the pathogenesis is explained by the release of different cytokines and chemokines. Conditions like septicemia, polytrauma, reperfusion injury closely mimics the pathophysiology of acute pancreatitis although it has nothing to do with the release of pancreatic zymogens. Damage to the cells of the acini propagates a series of catastrophic events. The first safeguarding mechanism includes limiting the area of inflammation. Failure of this protective strategy promulgates a cascade of catastrophic

events ultimately leading to Systemic Inflammatory Response Syndrome. Systemic Inflammatory Response Syndrome eventually boils down to acute respiratory response syndrome, hypotension, acute kidney injury and Multi Organ Dysfunction Syndrome. Events that trigger progression from a contained inflammation to a systemic dysfunction are remarkably unclear.

In conclusion, severe acute pancreatitis progresses from a locally controlled pancreatic inflammation to a multi systemic derangement. Systemic Inflammatory Response Syndrome is defined as two or more of the following, hypo or hyperthermia (<36 or >38°C), tachycardia (>90 bpm), increased respiratory rate (>20 pm, or CO_2 <4.3 kiloPascals), leucopenia or leukocytosis (<$4x10^3$ or >$12x10^3$ cells/dL, or >10 % leucoblasts). At the other end of the spectrum, severe acute pancreatitis is complicated by a pauci-immune stage popularly known as conteractive inflammatory response syndrome. During this stage, the individual is more prone for infective process primarily from gut bacteria translocation thereby culminating in multiorgan dysfunction syndrome frequently complicated by poorly guided surgical intervention. The disease pathology is associated with significant hypovolemia primarily due to fluid loss into the third space, capillary leak and loss of systemic vascular resistance. Hypovolemia leads to anemic, hypoxic hypoxia and

hypotensive shock which can cause ischemia of the myocardium, cerebral and intestinal circulation and theoretically trigger microbial translocation from the gut lumen. All these events culminate in the florid production of systemic inflammatory mediators including various cytokines and chemokines. It is incompletely understood why only some patients progress onto florid forms of the disease while others experience only mild disease.

EPIDEMIOLOGY

The prevalence of acute pancreatitis in various studies ranges from 12 to 38 per 100000 population[1]. This variation can be explained by the differences in consumption of ethanol and in the prevalence of cholelithiasis in various parts of the world.

In our institution, acute pancreatitis accounts for 4-5% of daily hospital admissions in the surgical department. It also accounts for about 28% of the patients admitted as ‘acute abdomen’. Amongst the causes, alcohol accounts for nearly 94% of the cases, with gallstones accounting for 5% and post-ERCP, drug-induced pancreatitis and idiopathic causes contributing to less than 1%.

DEFINITIONS

ACUTE PANCREATITIS

Acute reversible inflammatory process of the pancreas with variable involvement of peri-pancreatic tissues and/or remote organ systems associated with increased serum levels of proteases. It is classified as either Interstitial pancreatitis (focal or diffuse pancreatic involvement with parenchymal tissue enhancement that might be either homogenous or a bit heterogenous on intravenous contrast) or Necrotising pancreatitis (diffuse or focal areas of non-enhancing parenchyma along with fat necrosis of the peripancreatic tissue. Criteria includes non-enhancing pancreas less than 50HU involving 30% or 3 cm of the pancreatic parenchyma.

PERIPANCREATIC FLUID COLLECTION

Fluid that extravasates out of the pancreas and collects into the lesser sac, anterior para-renal spaces and into the para-colic gutters. Can occur both in interstitial and necrotizing pancreatitis.

PSEUDOCYST OF PANCREAS

Collection of pancreatic secretions covered by granulation tissue wall occurring after a latency period of atleast four weeks from the onset of acute pancreatitis

MILD ACUTE PANCREATITIS

Pancreatitis that is associated with minimal organ dysfunction and an uneventful recovery.

MODERATELY SEVERE ACUTE PANCREATITIS

Pancreatitis associated with local complications and transient organ failure (< 48 hours)

SEVERE ACUTE PANCREATITIS

Pancreatitis associated with persistent organ failure and / or death. Local complications includes necrosis, abscess or pseudocyst. Organ failure includes shock, pulmonary insufficiency, renal failure or gastrointestinal bleeding.

Modified Marshall score

Table 1 Modified Marshall scoring system for organ dysfunction

	Score				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	≤101
Renal*					
(serum creatinine, μmol/l)	≤134	134–169	170–310	311–439	>439
(serum creatinine, mg/dl)	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular (systolic blood pressure, mm Hg)†	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH<7.3	<90, pH<7.2

For non-ventilated patients, the FiO_2 can be estimated from below:

Supplemental oxygen (l/min)	**FiO_2 (%)**
Room air	21
2	25
4	30
6–8	40
9–10	50

A score of 2 or more in any system defines the presence of organ failure.
*A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 μmol/l or ≥1.4 mg/dl.
†Off inotropic support.

CLASSIFICATION OF ACUTE PANCREATITIS

Atlanta* criteria (1992)

- Mild acute pancreatitis (80% cases)
  (Acute Interstitial/edematous pancreatitis)
  - Acute Absence of organ failure
  - Absence of local complications

- Severe acute pancreatitis(20 % cases)
  (Acute Hemorrhagic Necrotizing (fulminant) pancreatitis)
  - Local complications **+/-**
  - Organ failure defined as
    - SBP < 90 mm Hg
    - $PaO_2 \leq 60$ mm Hg
    - GI bleed ≥ 500 ml/24 hrs
    - Cret ≥ 2 mg/dL after rehydration
  - Ranson score ≥ 3 or APACHE ≥ 8

Revised Atlanta criteria (2012)

- Mild acute pancreatitis
  - Absence of organ failure
  - Absence of local complications

- Moderately severe acute pancreatitis
  - Local complications **+/-**
  - Transient organ failure(<48 h)

- Severe acute pancreatitis
  - Persistent organ failure**(>48 h) and/or death

*defined as a score of 2 or more for one of these(CVS, Renal, Resp) organ systems using the modified Marshall scoring system

DIAGNOSIS

Diagnosis of pancreatitis is based on clinical features, biochemical investigations and imaging studies.

CLINICAL FEATURES

The dominant symptoms of pancreatitis include pain in the epigastric region, nausea, and vomiting. Typically the pain is located in the epigastric or peri-umbilical region, but it may also involve both upper quadrants, the lower chest and the lower abdomen. It can have a pleuritic component and therefore radiate to one or both the shoulders. The pain is described as being knife-like and radiating to the back. Usually abrupt in onset, it slowly increases in magnitude to reach a maximum level. The pain is usually constant, however it is mitigated by leaning forward or lying on one side with the knees drawn upward. The vomiting may lead to gastro-esophageal tears popularly known as Mallory-Weiss syndrome and upper GI bleeding. Eventhough Ryle's tube can temporarily improve the symptoms of vomiting and retching, the pain does not fully subside even after decompression of the stomach. Dehydration, poor skin turgor, tachycardia, hypotension, and dry mucous membranes are the common signs seen.

PHYSICAL FINDINGS

The clinical findings depends on the severity of acute pancreatitis. With mild disease process, the abdomen may be normal or reveal only mild epigastric tenderness. However, with severe pancreatitis, significant abdominal distention associated with generalized guarding and abdominal rigidity might be present. The nature of the pain need not always correlate with the clinical findings or the extent of pancreatic inflammation. Patients are frequently noted to be rolling or moving around to find a more comfortable position which apparently decreases their abdominal pain and this might help differentiate patients with a perforated hollow viscus who often remain motionless because even the slightest of movements deteriorates their abdominal pain. Patients with severe pancreatitis usually appear anxious and morbid. Hyperthermia, usually recognized, is due to the release of pro-inflammatory cytokines and chemokines, from the inflamed pancreas. Hypovolemia can cause hypotension, tachycardia and tachypnea and the dreadful triad of acidosis, hypothermia and coagulopathy can be fatal. Other findings include collapsed neck veins, dry skin, and diminished subcutaneous elasticity and dry mucous membranes. Abdominal pain makes breathing difficult

which leads to diminished breath sounds in the lower lung fields and subsequent atelectasis may be present.

A pleural effusion is seen commonly on the left side but may involve the right side in extensive pathology involving the pancreatic head. Patients with severe pancreatitis frequently develop adult respiratory distress syndrome and not rarely, patients with pancreatitis have altered mental status due to the CNS changes induced by alcohol per se, or hypotension, hypoxemia and release of toxic cytokines due to the pathophysiology of acute pancreatitis. In biliary pancreatitis, jaundice may reflect distal common bile duct obstruction by stones, but jaundice can also occur in any type of pancreatitis due to the ductal obstruction by the injured pancreas or due to cholestasis induced by the severity of the illness itself. The abdomen may become distended and tympanitic and bowel sounds absent as a result of paralytic ileus. A mass in the epigastric region, indicating the inflamed pancreas and the surrounding peri-pancreatic collections, can be present. Rarely retroperitoneal hemorrhage seen during severe acute pancreatitis can cause Grey Turner's sign (flank ecchymoses) or Cullen's sign (peri-umbilical ecchymoses). Occasionally, severe AP causes subcutaneous fat necrosis that present as tender subcutaneous induration and erythema resembling erythema nodosum.

Acute pancreatitis is diagnosed by the presence of two or more of the following features namely severe epigastric pain, amylase or lipase levels more than 3 times higher than reference level and CECT abdomen demonstrating findings of pancreatic inflammation. Computed Tomography is not quintessential for the diagnosis of acute pancreatitis as in the earlier phase of the disease (within 72 to 96 hrs of the onset of disease), a contrast enhanced CT scan often fails to delineate features of peri-pancreatic collections and necrosis as the disease process is a dynamic one. However, in patients presenting several days after the onset of pain in the abdomen, where the serum amylase and lipase levels might have become normal or in patients with organ dysfunction of unknown etiology, CT scan plays an important role in establishing the diagnosis.

IMAGING

The imaging modality at admission should include a chest and an abdominal roentgenogram that demonstrates ARDS, pleural effusion and free intra-peritoneal air under the diaphragm (which can sometimes be misleading as it may be due to the presence of anaerobic gas forming organisms). This is usually followed by a trans-abdominal ultrasonogram which is very sensitive to detect GB or CBD sludge and the presence of cholelithiasis thereby presuming a diagnosis of biliary pancreatitis. However, in the absence of gallstones or GB sludge, a dilated CBD (>8

mm if the patient is 75 years or younger; >10 mm if the patient is more than 75 years) or the elevation of serum ALT greater than 100 U/L and ALT level greater than AST level strongly suggests that acute pancreatitis is of biliary origin. These levels provide good guidance for the diagnosis of biliary pancreatitis but must be stressed that they are not absolute cutoff points. If the etiology still remains elusive, then EUS can be used to detect cholelithiasis, GB and CBD sludge. Contrast enhanced CT is often used to demonstrate peri-pancreatic fluid collections and pancreatic parenchymal or peri-pancreatic fat necrosis. A contrast enhanced CT scan has to be done in patients who fail to improve even after seven to ten days after the onset of symptoms. The terminology for describing pathologic changes in and around the pancreas has seen numerous variations in the revised Atlanta classification. A common mistake while reporting peri-pancreatic collections is to refer it as 'pseudocys*t*' for a homogeneous peri-pancreatic fluid collection that contains fluid and considerable amounts of necrotic pancreas. This pitfall arises as the CECT fails to discriminate solid necrotic components within a collection that is predominantly fluid. MRI or USG abdomen can circumvent this by means of demonstrating the presence or absence of necrosis in such apparently homogenous collections. The absence of necrosis is a necessity to be called a 'pseudocyst'.

SEVERITY CLASSIFICATION

The updated Atlanta classification overcomes the shortcomings of its predecessor published in 1992 due to the improved understanding of the pathophysiology of acute pancreatitis, and it includes, a sub-division into three types such as *mild, moderately severe* and *severe* acute pancreatitis. In the early phase of the disease process, this category is dependent on clinical parameters, and in the following period, the other subdivision is dependent on a combination of clinical parameters as well as morphologic complications which increase in-patient hospital stay, and necessitating active surgical, radiological or endoscopic intervention and supportive management namely, the requirement for vasopressors, mechanical ventilation, or renal replacement therapy. The terms 'severe' and 'necrotizing' pancreatitis do not fully overlap. Necrotizing pancreatitis is defined as the presence of parenchymal necrosis or peri-pancreatic fat necrosis. The updated classification includes peri-pancreatic necrosis only (i.e., without necrosis of the pancreatic parenchyma) in the category of necrotizing pancreatitis. Edematous (interstitial) pancreatitis usually runs a mild clinical course, but a few of them suffer a fulminant course and die within 2 to 5 days and these patients clearly have severe pancreatitis, but are not classified as necrotizing one.

The clinical course of pancreatitis is extremely haphazard and it defies prediction. It may range from complete recovery within a few days to SIRS and MODS and mortality within a few hours or days. Though several predictive scores have been published to guide physicians in the initial treatment and the level of observation required in each individual, their value in everyday clinical practice is limited. If an individual meets a cutoff value of a particular predictive score, it only means that he can at that stage of the disease process, be classified as having ‘predicted severe pancreatitis’. The clinical value however, is limited, as the subset of patients truly progressing to severe acute pancreatitis (positive predictive value) is anywhere between 50% and 70%. However, the predictive scores also have a negative predictive value ranging between 85% and 90% and therefore are most useful in excluding patients at risk for severe pancreatitis, because of the fact that, patients with mild form of acute pancreatitis carry a 10% to 15% risk of developing the severe acute pancreatitis.

BISAP SCORE

BUN>25 mg/dL

Impaired mental status (Glasgow Coma Scale Score<15)

SIRS

SIRS is defined as two or more of the following:

(1) Temperature of <36 or >38°C

(2) Respiratory rate >20 breaths/min or PaCO2<32 mmHg

(3) Pulse>90 beats/min

(4) WBC <4,000 or >12,000 cells/mm^3 or >10% immature bands

Age>60 years

Pleural effusion detected on imaging

One point is assigned for each variable within 24 h of presentation and added for a composite score of 0-5.

Modified Glasgow/PANCREAS score

- **P**a**O2** < 8kPa (60mmhg)
- **A**ge > 55 years
- **N**eutrophils: **WBC** >15 x109/l
- **C**alcium < 2mmol/l
- **R**enal function: (**Urea** > 16mmol/l)
- **E**nzymes: (AST/ALT > 200 iu/L or **LDH** > 600 iu/L)
- **A**lbumin < 32g/l
- **S**ugar: (Glucose >10mmol/L)

***Applicable for both** gallstone and alcohol induced pancreatitis within 48 hours of admission

***Omission of age/serum transaminase** increases the predictive valve of scoring system as serum transaminase did not differ significantly between mild and severe pancreatitis

*Bold 4 factors are **independently significant** in predicting the severity

APACHE II SCORE

Physiologic Variable	High Abnormal Range					Low Abnormal Range				
	+4	+3	+2	+1	0	+1	+2	+3	+4	Points
Temperature - rectal (°C)	≥41°	39 to 40.9°		38.5 to 38.9°	36 to 38.4°	34 to 35.9°	32 to 33.9°	30 to 31.9°	≤29.9°	
Mean Arterial Pressure - mm Hg	≥160	130 to 159	110 to 129		70 to 109		50 to 69		≤49	
Heart Rate (ventricular response)	≥180	140 to 179	110 to 139		70 to 109		55 to 69	40 to 54	≤39	
Respiratory Rate (non-ventilated or ventilated)	≥50	35 to 49		25 to 34	12 to 24	10 to 11	6 to 9		≤5	
Oxygenation: A-aDO2 **or** PaO2 (mm Hg) a. FIO2 ≥0.5 record A-aDO2 b. FIO2 <0.5 record PaO2	≥500	350 to 499	200 to 349		<200 PO2>70	 PO2 61 to 70		 PO2 55 to 60	 PO2<55	
Arterial pH (preferred) Serum HCO3 (venous mEq/l) (not preferred, but may use if no ABGs)	≥7.7 ≥52	7.6 to 7.69 41 to 51.9		7.5 to 7.59 32 to 40.9	7.33 to 7.49 22 to 31.9		7.25 to 7.32 18 to 21.9	7.15 to 7.24 15 to 17.9	<7.15 <15	
Serum Sodium (mEq/l)	≥180	160 to 179	155 to 159	150 to 154	130 to 149		120 to 129	111 to 119	≤110	
Serum Potassium (mEq/l)	≥7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5	
Serum Creatinine (mg/dl) Double point score for acute renal failure	≥3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4		<0.6			
Hematocrit (%)	≥60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20	
White Blood Count (total/mm3) (in 1000s)	≥40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1	
Glasgow Coma Score (GCS) Score = 15 minus actual GCS										
A. Total Acute Physiology Score (sum of 12 above points)										
B. Age points (years) ≤44=0; 45 to 54=2; 55 to 64=3; 65 to 74=5; ≥75=6										
C. Chronic Health Points (see below)										
Total APACHE II Score (add together the points from A+B+C)										

MODIFIED BALTHAZAR – CT SEVERITY INDEX

Chart 1 CT morphological index and CT severity index for acute pancreatitis.

Inflammatory process – Balthazar's morphological index for acute pancreatitis		
Grade	Tomographic finding	Scoring
A	Normal pancreas.	0
B	Focal or diffuse pancreatic enlargement.	1
C	Pancreatic alterations associated with peripancreatic inflammation.	2
D	Single fluid collection.	3
E	Two or more fluid collections and/or presence of gas within the pancreas or within peripancreatic inflammation.	4
Pancreatic necrosis		
Tomographic finding		Scoring
Absence of necrosis.		0
< 30% necrosis.		2
30% to 50% necrosis.		4
> 50% necrosis.		6

– Ranson's Prognostic Criteria

NON-GALLSTONE PANCREATITIS	GALLSTONE PANCREATITIS
At Admission	
Age >55 yr	Age >70 yr
White blood cells >16,000/mm^3	>18,000/mm^3
Blood glucose >200 mg/dL	>220 mg/dL
Serum lactate dehydrogenase >350 IU/L	>400 IU/L
Serum aspartate aminotransferase >250 IU/L	>250 IU/L
During Initial 48 hr	
Hematocrit decrease of >10 %	>10%
Blood urea nitrogen increase of >5 mg/dL	>2 mg/dL
Serum calcium <8 mg/dL	<8 mg/dL
Arterial po_2 <60 mm Hg	NA
Serum base deficit >4 mEq/L	>5 mEq/L
Fluid sequestration >6 L	>4 L

The following table depicts the commonly performed predictive scoring systems in acute pancreatitis. Also included is their cutoff value in predicting acute severe pancreatitis

PREDICTIVE SCORE	**CUTOFF**
Modified Glasgow (or Imrie)	≥3 in first 48 hours
APACHE II	≥8 in first 24 hours
BISAP	≥3 in first 24 hours
Ranson	≥3 in first 48 hours
C reactive protein	>150 U/L in the first 72 hours
BUN during admission	>60 millimol/L

USUAL COURSE OF THE DISEASE PROCESS

Typically, pancreatitis during the acute stage, demonstrates a biphasic course. The initial (1^{st}) phase, lasting nearly two weeks is characterized by a SIRS (systemic inflammatory response syndrome). The second phase, CARS (counteractive anti-inflammatory response syndrome) is characterized by a state of immune-suppression[3]. Organ failure in the first phase (i.e. SIRS phase) is considered to be due to severe systemic

inflammation and is unrelated to infection. On the other hand, organ failure in the second phase (i.e. CARS) is occurs due to secondary infections, like infected necrosis probably arising due to the translocation of bacteria across the gut wall[4]. However, infections, do happen in the first (SIRS) phase, but ventilator associated pneumonia and bacteremia are the most common types. This finding is based on a study, which included a series of patients suffering from severe acute pancreatitis, where, it was deduced that these infections (ventilator associated pneumonia and septicemia) were most often diagnosed in the first week of admission. The respiratory and the cardio-vascular systems are the dominant systems involved in organ failure. The gastro-enteric system, suffering from a state of low flow and systemic inflammatory response syndrome, is difficult to figure out due to vague signs and symptoms, in comparison to indicators such as oxygen exchange, blood pressure, and urine output, which are well defined. In the first (SIRS) phase, organ failure may manifest rarely at admission itself, but is commonly elucidated at a median of two days after admission to the hospital. Nearly half of the mortality from severe acute pancreatitis is due to multi-organ failure and not infected necrosis, contrary to popular belief. This statement is strengthened from a series of cohort studies reviewed systematically, which concluded that 32% of patients who develop multi-organ dysfunction eventually succumb to the disease. Death occurring in

patients with concurrent multi-organ dysfunction and infected pancreatic and peri-pancreatic necrosis was 43%. The clinical course of severe acute pancreatitis, being highly variable, there may be a continuum between the systemic inflammatory response syndrome and counteractive antiinflammatory response syndrome phases.

The pathophysiology of severe acute pancreatitis is divided into the following three stages for a better understanding of the disease process.

- Week 1: Early onset organ dysfunction, SICU management, followed by improvement with supportive measures and continued ICU treatment[5] (Weeks 2 through 3).
- In the weeks to follow (weeks 3 through 5), clinical deterioration occurs. This sequence of events is highly indicative of necrosis getting infected. Without early organ failure, clinical stability is suddenly complicated by deterioration in weeks 3 through 4 of admission. Again, the probability of infected necrosis being the underlying etiology of clinical deterioration are high.
- Early onset organ dysfunction doesn't improve, even after 2 to 3 weeks of conservative treatment in the SICU management. At this stage, a fine-needle aspiration of one of the collections can help differentiate between persistent systemic inflammatory response syndrome and infected necrosis and therefore, determine the need

for intervention such as percutaneous drainage or necrosectomy. However, if, contrast enhanced CT scan shows air in the necrotic components, no further diagnostic procedures are required, and intervention to treat the source of infection is necessary.

TREATMENT OF ACUTE PANCREATITIS

CONSERVATIVE MANAGEMENT

First Phase (Systemic Inflammatory Response Syndrome) Treatment:

1. Adequate fluid resuscitation
2. Adequate pain relief.

- A fluid regimen guided by diuretics like frusemide (target output -1 mL/kilog/hr) is required during the first phase, until there is no evidence of organ failure[5].
- Careful monitoring and intravenous fluid supplementation in the initial twenty four hours of severe acute pancreatitis is most important
- As high as 20 litres of crystalloid solutions might be required. However, unrestricted fluid resuscitation may be rapidly fatal
- A recent RCT from China proved that very high fluid resuscitation aimed at keeping hematocrit less than 35% over the first forty eight hours was associated with increased mortality. In this initial phase

of the disease process, there is no role for any surgical, radiologic or endoscopic intervention for the necrotic components.

Second phase (Counteractive Anti-inflammatory Response Syndrome) treatment

- If the patient does not improve or deteriorates after initial stabilization, infection of the pancreatic and peri-pancreatic collections should be considered. Fine needle aspiration (FNA) of the necrotic collections and subsequent gram staining might be considered to confirm this.
- However, the possibility of false negative results and the risk of introducing infection remains a deterrent. Moreover, in cases of clinical deterioration, a negative FNA should not deter any kind of intervention.
- A randomized controlled trial based on Sweden demonstrated that intervention in individuals with acute necrotizing pancreatitis dependent on clinical deterioration rather than on routine fine needle aspiration results, indicated, that 92% of patients had infected necrosis confirmed at the time of surgical or percutaneous catheter intervention.
- However, presence of gas bubbles in peri pancreatic collections are considered as definitive evidence for infected necrosis that would

warrant an intervention (either image-guided or surgical) and fine needle aspiration is not indicated.

PREVENTION OF INFECTION

- In patients with acute severe pancreatitis, various prophylactic regimens have been explored for infection prevention because of its association with high mortality. Intestinal bacteria are considered responsible for the source of these infections and the present understanding is that these pathogens pass through the mucosal barrier in the first twenty four hours of disease process.
- In a recent multicentric trial, of all the infections diagnosed during the course of acute severe pancreatitis, usually, a septicemia or VAP was diagnosed at a median of eight days after the onset of epigastric pain, in the first instance.
- Infection of necrotic components was only deducted at a median of twenty-six days. Mortality from each infection including pneumonia and bacteremia was thirty percent
- Bacteremia (due to any cause) increased the risk of infection of pancreatic and peri pancreatic necrotic components from anywhere between 38% and 65%. This finding was strengthened in multivariate analysis which, demonstrated that persistent organ

failure (odds ratio 18) and bacteremia (odds ratio 3.4) were the strongest predictors of mortality due to severe acute pancreatitis

- Systemic IV antibiotics, early enteral feeding (oral or nasojejunal), selective bowel decontamination, and probiotics have been attempted to decrease the infectivity rates.
- Early enteral feeding is predicted to reduce small bowel microbial over-growth and to improve intestinal mucosal barrier function, thereby reducing bacterial translocation and subsequent infectious complications[7, 8]. A recent RCT by Daniel Bimmler et al. stated that, in patients with mild pancreatitis, enteral feeding can be started on the day of admission or the next day itself. In predicted severe acute pancreatitis, early enteral nutrition by nasojejunal (Freka tube) feeding tube within a span of 3 days can be started, if the patient cannot tolerate an oral diet[9].
- A recent meta-analysis confirmed that, in individuals diagnosed as severe acute pancreatitis, early enteral feeding decreases both infections complications and mortality when compared with TPN.
- There is not enough evidence to recommend between various enteral nutrition formulations, including glutamine supplementation.
- The best mode for administering enteral feeding via a nasogastric or a nasojejunal Freka tube is not universally accepted. Two RCTs,

each including nearly eighty cases did not find any difference in compliance for feeding and complications between nasogastric and nasojejunal routes[8]. These studies also failed to show relevant differences regarding various complication rates, such as vomiting, aspiration etc.

Systemic Antibiotics:

- Several reports have stressed the requirement for antibiotic prophylaxis in lowering the problems arising due to infections in acute severe pancreatitis. The initial trials showed somewhat positive effects. In the recent years, however, placebo controlled trials have failed to show a reduction of infections complications and mortality. A multicentric study from Germany demonstrated an increased prevalence of antibiotic resistance and a rise of fungal infections[10, 11].
- As a result, based on current evidence, there is no role for the routine prophylactic use of systemic intravenous antibiotics.

Selective Bowel Decontamination:

- As the intestine is postulated to be the source of bacterial flora for the infectious complications in acute severe pancreatitis, it is only a rational approach to administer enteral antibiotics.

- However, only one randomized control trial studied the efficacy of Selective Bowel Decontamination in acute severe pancreatitis and compared the effect of enteral antibiotics such as norfloxacin, colistin and amphotericin versus placebo[12]. A decrease in death rates in the Selective Bowel Decontamination group, primarily due to a decrease of gram(-) bacteria of necrotic components was found[13]. However, this trial has not been reproduced, and this SBD strategy has not been universally accepted. However the collected material indicates that the idea of early interventional treatment in the wide spectrum of the disease process involving enteric microbial overgrowth, mucosal breakdown leading to translocation of gut microbia and subsequent septicemia warrants detailed study.

Pro-biotics:

- Many multicentric studies have deduced that prophylactic administration of pro-biotics can decrease the complications arising due to infections in pancreas and liver surgery.
- A positive effect of prophylactic pro-biotics use in severe acute pancreatitis is shown in 2 randomized control trials from Europe. On a contrast note, based on a pro-biotics trial in Holland, it was demonstrated that, no benefit was gained in individuals with severe acute pancreatitis but a 2.5fold higher

mortality rate occurred in the individuals receiving probiotics[13]. Hence, no proper guideline exists at this point of time, despite several follow up studies, clinically and experimentally. Therefore, at this point of time, prophylactic enteral probiotics are not indicated in cases with predicted acute severe pancreatitis.

INTERVENTIONAL TREATMENT

First Phase (Systemic Inflammatory Response Syndrome) Treatment

- Intervention in the first phase of severe acute pancreatitis aims at managing acute life-threatening organ dysfunction and prevention of further deterioration. As per existing evidence, the only method to prevent further deterioration in acute pancreatitis is Endoscopic Retrograde Cholangio Pancreatography -ERCP and sphincterotomy, even though its exact place in the management armamentarium is yet to be established[18, 19].
- In 1989, a trial on early surgical necrosectomy, in first phase of the disease process, was performed, where, ‘early’ surgical treatment within seventy-two hours of the onset of abdominal pain was compared with ‘late’ operation after twelve days. However, the study group prematurely terminated the trial due to a much higher (though statistically insignificant) mortality rates for ‘early’

surgical intervention group (58% vs. 27%). In accordance with this trial, early surgical intervention to debride necrotic components was essentially given up[14].

- It was universally accepted that in the initial stage of the disease process, the pathophysiology is dominated by systemic inflammatory responses rather than by the presence or absence of infection of necrosis. Therefore, there is no apparent benefit is to be expected from early surgical intervention, if removal of infected necrosis is the sole indication for surgery[16, 17].

Indications for Acute Interventions:

- The following complications form the basis for early surgical intervention. They are,

1. Abdominal compartment syndrome[15]
2. Bowel ischemia
3. Hollow viscus perforation
4. Severe bleeding, not amenable to angiographic coiling.

- The definition of abdominal compartment syndrome, includes, IAP greater than 20 mm of mercury with features of new-onset organ dysfunction such as hypotensive shock, decreased urine output, respiratory distress, etc..

- Even though the optimum management of ACS is not fully standardized, a consensus meeting suggested that percutaneous catheter drainage serves as an initial stabilisation method to drain intra-abdominal collection, if significant[15]. If PCD failed to lower the pressure immediately or if there is no residual fluid to be drained, and the syndrome persists, then emergency laparotomy for decompression is advised.
- The pancreas, however, should not be explored at this stage because it is very early to drain the necrotic debris in a safe manner, and for the disadvantage of contaminating the necrotic debris.
- Early Endoscopic Retrograde Cholangio Pancreatography with sphincterotomy in gall stone pancreatitis: The present understanding of the pathogenesis of biliary pancreatitis is that a gallstone, released from the GB into the CBD, causes transient blockage of the ampulla of Vater, thereby leading to obstruction of the pancreatic duct and secondary damage to the exocrine cells with auto-digestion of the exocrine pancreas due to the extravasation of proteases from the pancreatic juice[18, 19]
- Therefore, early relief by Endoscopic Retrograde Cholangio Pancreatography with sphincterotomy should end this process at an early stage and mitigate the risk of progression to complications.

However, a recent meta-analysis demonstrated that there is no overwhelming benefit of routine ERCP with sphincterotomy in patients with predicted acute severe biliary pancreatitis in the absence of evidence of cholangitis.

- On this aspect, a recent prospective multi-center trial concluded that sphincterotomy via ERCP decreases the severity and halts the pathogenesis of the disease process in individuals with severe gallstone pancreatitis and cholestasis (arbitrarily defined as bilirubin levels >2.3 millig/dL [>40 micromol/L] or age adjusted dilated CBD diameter) [19].

Second Phase (Counteractive Anti-inflammatory Response Syndrome) Treatment:

- Management of infected necrosis during this second (CARS) phase, the patient experiences a second attack of septicemia, this time, occurring due to a secondary contamination of peri-pancreatic necrosis via translocation of gut microbes.
- Therefore, documented or suspected infection of pancreatic or peri-pancreatic necrosis with signs of sepsis is the most accepted indication for intervention, either radiologic, endoscopic, or surgical.

- However, rare requirements for interventional treatment include ACS, severe bleeding, GOO, common bile duct obstruction, and hollow viscus perforation.
- Once a decision for interventional treatment is taken, the options include laparotomy with necrosectomy, minimal access surgery, endoscopic, and image guided procedures.
- The incidence and advantages of minimal access procedures is exponentially increasing. However, its correct position and requirement in the treatment algorithm for all these procedures is not exactly formulated till date. Our study tries to highlight the role of PCD technique in the treatment strategy of this phase of the disease process.

Timing of Intervention:

- An experienced multi-disciplinary group should help decide 'which', 'when', and 'where', a particular procedure must be utilized. Postponement of interventional treatment until the parenchymal and peri-pancreatic collections become organized which occurs about 4 weeks after the onset of disease, is beneficial[6, 21].
- These so called encapsulated collections are referred to as 'walled off pancreatic necrosis (WOPN)'. In the emergency clinical

situations described earlier, encapsulation of the peri-collection might not have been completed, but clinical deterioration would warrant an emergency intervention

- Administration of antibiotics to allow for further encapsulation, under close guidance of the clinical developments and periodic contrast enhanced CT scan, performed at regular intervals, to prevent septicemia, is a valid alternative to postpone surgical intervention.
- In the multicentric trial mentioned earlier, necrosectomy by open approach was done after a median period of twenty seven days, and the fatality rate was 25%. If intervention was performed in the first 2 weeks, mortality was 75%.
- Based on the findings described above, postponement of interventional treatment until about four weeks is the preferred management strategy. The length of the conservative management interval is mainly determined by the completeness of encapsulation of peri-pancreatic collections and the clinical condition of the patient. This strategy is therefore, only applicable to the subset of patients who survive the first phase of systemic inflammatory response syndrome and develop infection of necrosis with signs of septicemia, in the second phase of counteractive inflammatory response syndrome.

Types of Intervention

Percutaneous Catheter Drainage:

- Percutaneous Catheter Drainage (PCD) using pigtail catheter is the least invasive procedure in the treatment of infected pancreatic and peri-pancreatic necrosis. This catheter can be placed percutaneously through the retroperitoneum (commonly through the left flank) or trans-abdominally, but also across the lumen of the stomach and/or the small intestine (mostly duodenum), called as 'trans-luminal' approach.
- A recent systematic review[21] suggested that nearly 55% of patients with acute severe pancreatitis, percutaneous pigtail catheter drainage can be the sole intervention needed for cure. In this review, in about 99% of the cases, the procedure was a success technically. The pre-operative organ dysfunction was present in 77% of the patients, and the case fatality rates was 17%.
- Accordingly, a multicentric trial from the far West such as the United States and Canada demonstrated that 25% of the cases with infected necrotizing pancreatitis can be solely managed by a PCD procedure.
- Finally, a landmark multicentric trial from Europe concluded that PCD is possible in 99% of the cases. In patients who do not

improve after adequate drainage and lavage through the catheter, necrosectomy should be considered as the obvious alternative. The percutaneous pigtail catheter can be used as a roadmap for minimally invasive necrosectomy. This two-step approach, PCD as the initial step, following which a catheter-guided minimal access necrosectomy as the next step, is popularly known as the "step-up" approach and is now believed to be the standard of care in patients with infected necrosis[6].

Minimally Invasive Necrosectomy - The most frequently performed minimal access surgery is the VARD procedure known as video-assisted retroperitoneal debridement[21, 23].

- The initial step of this treatment strategy involves placing a left sided percutaneous catheter retroperitoneally through the left flank if the collection can be reached through this route. The patient is positioned supine with the left side up. Using the pigtail catheter as a guidance, a 5 to 7 cm incision is made, and the necrotic collection is reached. The initial pus and necrosis are removed in a blind manner.
- This is followed by introduction of a zero degree laparoscope to remove all the necrosis in reach, under direct vision. Only loosely attached necrotic debris are sucked out and lavaged to reduce

bleeding risks. It is seldom necessary to get rid of all necrotic debris. In contrast to totally percutaneous necrosectomy techniques, VARD can help remove large pieces of necrotic debris[22].

- Generally, in a well-organized necrosis, necrosectomy can be performed safely without concomitant risk of hemorrhage. Following near-complete debridement, 2 wide bore drains (preferably intercostal drains) are kept into the empty cavity, one at the deepest point and the other more shallow.
- In the postoperative setting, continuous lavage using (2L, 4L and 6 L of) normal saline is performed through the drains in the first 3 days.
- In a recent Dutch multicentric RCT, in individuals with documented / suspected contamination of necrotic debris, the minimally invasive "step-up" approach was studied in comparison with primary open necrosectomy. The results were overwhelmingly in favor of the "step-up" approach, but there wasn't any significant difference in case fatality rates[6].
- Carter at al from Glasgow described a totally minimally invasive percutaneous retroperitoneal necrosectomy using an operating nephroscope. A recent series confirmed a decrease in mortality when using this technique compared with historical controls.

Endoscopic Transluminal Necrosectomy:

- If video-assisted retroperitoneal debridement is technically not feasible when the infected necrosis does not reach far enough left, then endoscopic necrosectomy either via trans-luminal or trans-gastric is a good alternative. Since the earlier description of the trans-gastric approach by Seifert in 2000, this technique has been increasingly adopted universally with success rates ranging from 80% to 93% and mortality from 0% to 6%[26].
- Controlled trials, however, are required, as selection bias may influence the results, and in many trials, the rate of infection of the necrotic components was low.
- The advantages, however, are that no abdominal incision is required, and subsequently, external pancreatic fistula is a rare occurrence, because, an internal fistula to the stomach or duodenum, is created. Incisional hernia, often difficult to treat after open necrosectomy, can also be avoided. But, this technique is not a one-time treatment and the need for repeated, multiple procedures to remove sufficient amounts of necrosis is a distinct disadvantage as for as the trans-luminal technique is concerned.

Open Necrosectomy:

- Before the conclusions of "PANTER" trial were published[6], primary 'early' open necrosectomy was considered as the standard of care in patients with suspected or confirmed contamination of pancreatic necrosis.
- The commonly performed surgical method of open necrosectomy includes laparotomy with placement of a retroperitoneal drainage tube following removal of debris mainly the 'fluid under pressure'.
- *Beger's Lavage* – Beger described placement of lavage systems in the lesser sac after surgical removal of the necrotic debris and lavaging with serially increasing levels (such as 2L, 4L, and 6 L per day) of normal saline (0.9%).
- Lavage serves various purposes including mechanical debridement, prevention of tube obstruction and dilution of pancreatic juice. The case fatality rate of this procedure is approximately 25%.
- Yet another technique of open procedures includes '*open necrosectomy and closed packing'*. Here necrotic debris is approached via the transverse mesocolon and debridement is performed in a blunt manner, with a target to remove all necrotic and particulate debris. Subsequently the resultant cavity is packed

with gauze-stuffed Penrose drains and its removal is performed on a day to day basis.

- Many surgeons perform an open abdominal approach where periodic, scheduled re-laparotomies is performed every 3 to 5 days. As the mortality of this procedure is approximately 70%, it is encouraged to use this procedure only as a ‘rescue’ technique as the last resort, when it is technically impossible to close the abdomen.

MATERIALS AND METHODS

PLACE OF STUDY:

Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-600003.

STUDY DESIGN:

Observational (prospective) study

PERIOD OF STUDY:

March 2016 to September 2016

INCLUSION CRITERIA:

Patients above 18 years of age admitted in RGGGH with acute necrotizing pancreatitis (defined as increase in serum amylase level within the first 48 hours, with a threshold of 3 or 4 times the upper normal range, and show evidence of pancreatic necrosis on the contrast-enhanced CT performed between the 48th and 72nd hrs after the onset of abdominal pain) and having one of the following conditions,

(i)Infected necrotic peripancreatitic collections

(ii)Symptomatic sterile peripancreatitic collections

EXCLUSION CRITERIA:

1. Patients with collections after pancreatic surgery

2. Patients who underwent treatment for pseudocyst, which is classified as a late (>4-6weeks) complication of pancreatitis

3. Those not willing to participate in the study

4. Patients below 18 years of age

5. Patients who are pregnant

SAMPLE SIZE:

35 patients {Sample size N=Z^2P(1-P)/d^2 where Z=1.96,P=10%,d=10%}

PROCEDURE:

This is a prospective study based on collected of 35 patients with severe acute pancreatitis managed at the Institute of General Surgery, Madras Medical College and RGGGH between March 2016 and September 2016. The diagnosis of acute pancreatitis is done based on clinical features, increased serum amylase or lipase levels and imaging criteria. The definition of severe acute pancreatitis is in accordance with the

modified Atlanta classification as modified Balthazar or CT severity index (CTSI) greater than 7.

On admission, all the cases are resuscitated with intravenous fluids, organ dysfunction management, analgesics and antibiotics such as imipenem/cilastatin or meropenem and metronidazole administered prophylactically. Within three days of admission, early nutritional resuscitation was taken care of. The enteral feeding route, whether per-oral, nasogastric or nasojejunal was decided by the performance status of the individual. In accordance with the culture and sensitivity of the drain fluid or blood and urine samples, appropriate antibiotics was administered. Continuous monitoring of all the cases were done round the clock to watch out for potentially life threatening complications like multi organ dysfunction and septicemia. Radiological diagnosis using usg of abdomen and/or CT was done in a periodic manner to help assess the localization or the spread of inflammatory collections and the development and delineation of necrosis.

Supportive treatment was offered to those individuals who showed improvement when compared with their status during admission. Those cases who developed (i) contaminated peripancreatic collections (ii) sterile but symptomatic (abdominal hypertension or compartment syndrome) collections (iii) infected necrosis, (iv) progressive organ

dysfunction underwent image-guided PCD or surgical necrosectomy. In cases with 5cm or greater collections, demonstrating unrelenting hyperthermia, elevated leucocytes and progressive organ dysfunction, an USG or CT-guided PCD was performed using a 12 Fr pigtail catheter by the Seldinger technique via the transperitoneal or retroperitoneal route avoiding injury to the bowel and other vital structures. This technique was preceded by Vit. K, FFP and platelet supplementation to treat coagulopathy, when required. PCD was anchored to drain by gravity and NS irrigation was performed when solid necrotic debris was drained or it caused blockage of the tube. Periodic ultrasonogram abdomen was performed to verify the emptying of the collections and the requirement to flush the tube and reposition the same. Periodic contrast enhanced CT scans were done, if need be, to monitor the resolution or the progression of the disease process and the requirement for surgical necrosectomy. If the drain quantity was lower than ten milliliter for 2 days, the PCD was removed under ultrasound guidance. PCD success was defined when there was reduction in the size of the fluid collections, control of septicemia and absence of requirement of surgery.

Surgery was performed if there was no clinical improvement, worsening organ failure in spite of PCD or when the individuals exhibited Warshaw's theory of un-wellness[37]. The surgery included open necrosectomy and a irrigation and drainage of the lesser sac. The timing

of the surgical treatment, no. of days of SICU care, duration of in-patient treatment and case fatality rates were noted.

STATISTICAL ANALYSIS

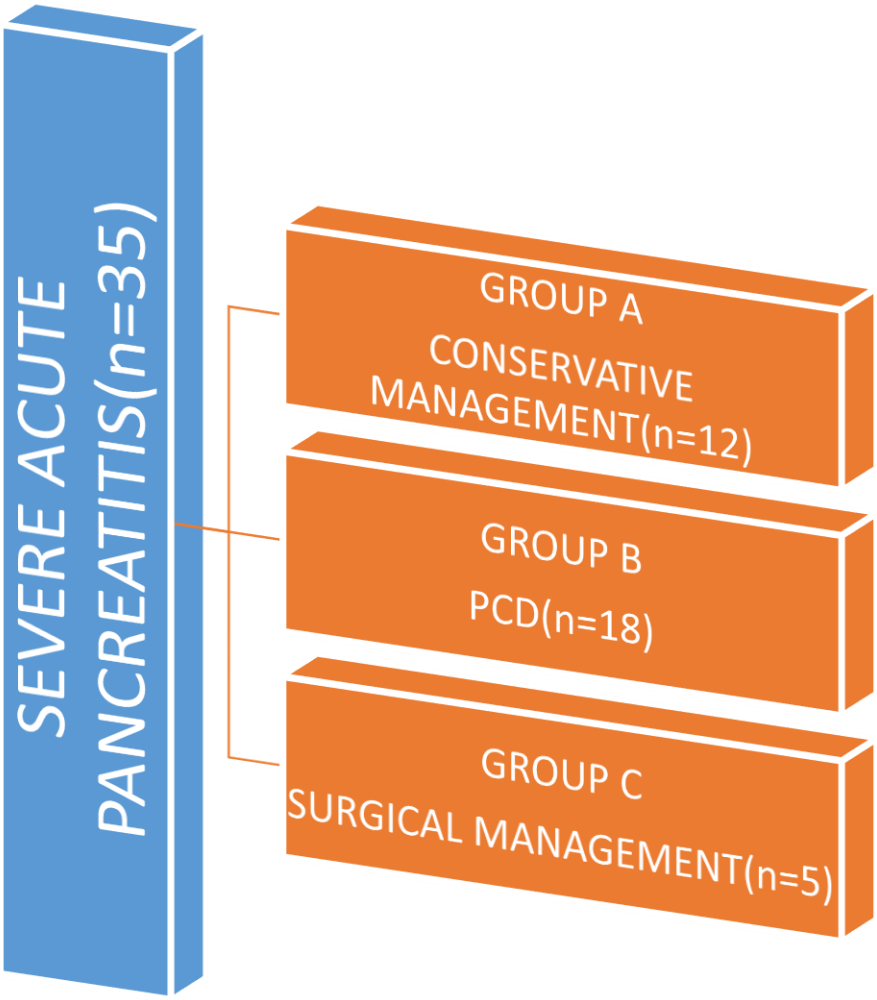
All the collected data were tabulated on MS Excel sheet. For the categorical and continuous data, the calculations were denoted by numbers and percentage of the total and mean ± S.D. respectively. Student T test and chi-squared tests were performed to calculate the continuous and categorical variables respectively using SPSS (Statistical Package for Social Science) software. A “P” value < 0.05 was considered as a statistically significant result.

OBSERVATION AND INFERENCE

- 35 consecutive patients admitted to the Institute of General Surgery, RGGGH with severe acute pancreatitis were studied.
- The etiology of severe acute pancreatitis were as follows, ethanol related in 32(91.4%) cases, gallstones in 2(5.7%) cases and idiopathic in 1 (2.9%) patient.
- Male patients constituted 31(88.6%) and females 4(11.4%) of the total study group.
- The age distribution of the patients were as follows- 4(11.4%) of age group 20 – 30 years, 13(37.1%) of age group 30 – 40 years, 12(34.3%) of age group 40 – 50 years, 6(17.1%) of age group 50 – 60 years. The mean and standard deviation of this age distribution was 40.71 ± 9.06.
- Serum amylase was elevated to more than three or four times the normal in 28 (80.0%) whereas lipase levels was increased in 31 (88.6%) cases.
- Acute kidney injury as suggested by elevated blood urea and serum creatinine levels was present in 6(17.1%) patients and contrast CT scans were promptly avoided in these patients. Those patients who

underwent CECT abdomen had instances of SAP with a modified Balthazar or CT severity index more than seven.

CATEGORIZATION OF PATIENTS



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- The patients were initially treated in the SICU; based on their response to conservative treatment, they were categorized into 3 divisions: first, patients manageable by SICU treatment alone; second, patients who underwent PCD and third, patients who underwent primary surgery or surgery due to the non-improvement of the conservative and PCD management.
- Of the 35 patients, 12(34.3%) patients were treated successfully by conservative treatment alone (Group A), whereas 18(51.4%) patients required PCD (Group B).
- Out of 18 cases in Group B, 12 patients (66.7%) were treated successfully and there was no necessity for surgical management (Group B1) whereas 2 (11.1%) patients were operated on (Group B2).
- Group C consisted of patients who underwent primary surgical management-5(14.3%) patients.

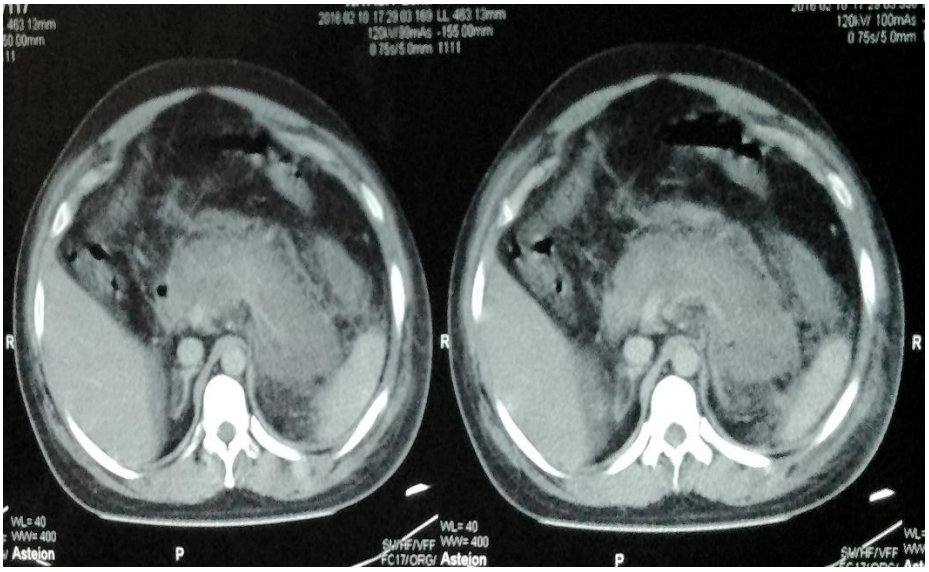


Fig.1. CONSERVATIVE MANAGEMENT

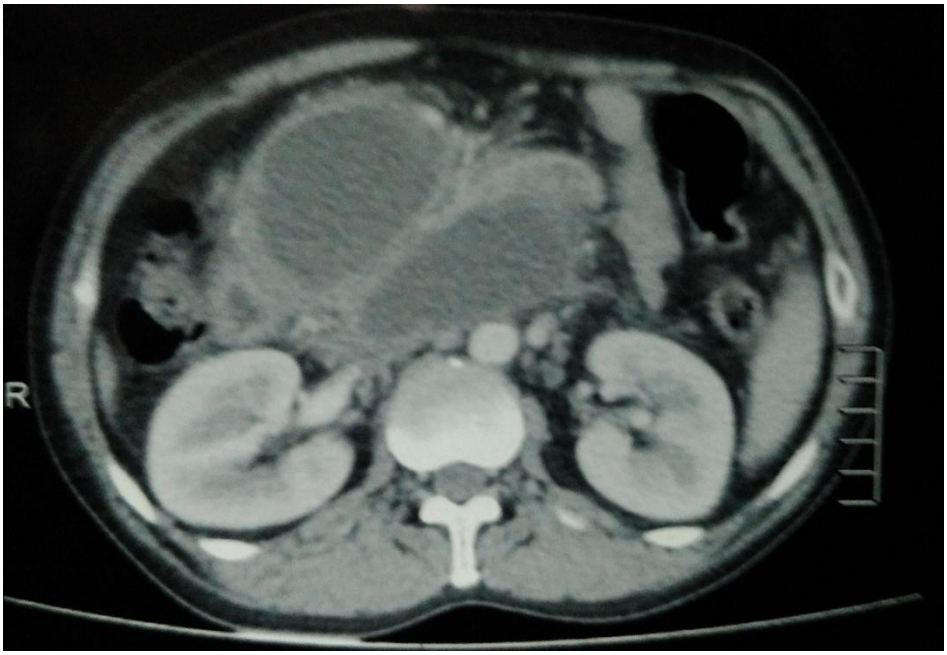


Fig.2. PERCUTANEOUS CATHETER DRAINAGE GROUP

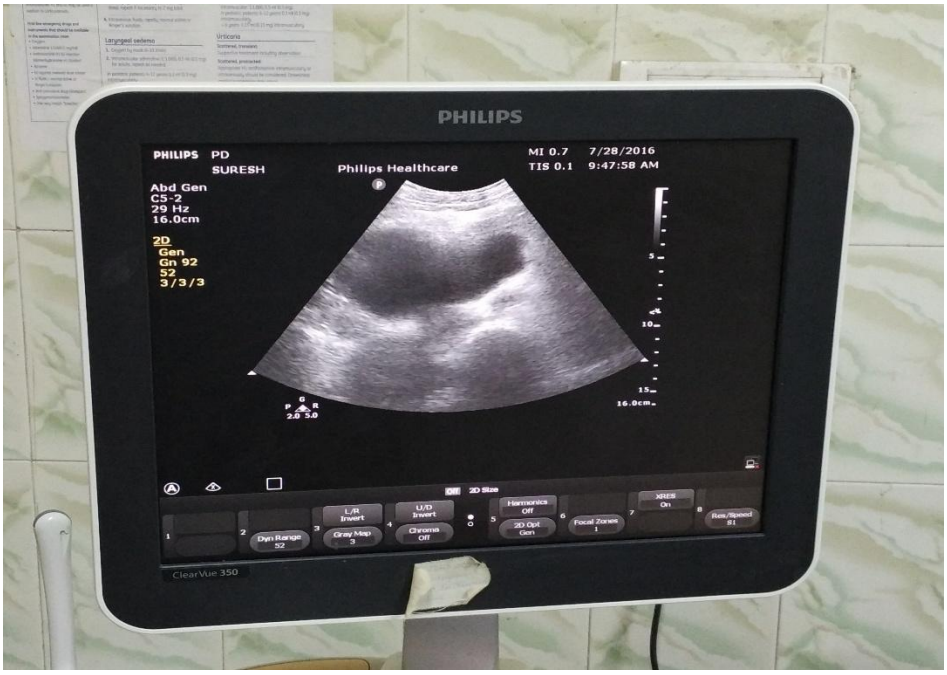


Fig.3. USG GUIDED PCD

Fig.4. DRAIN FLUID

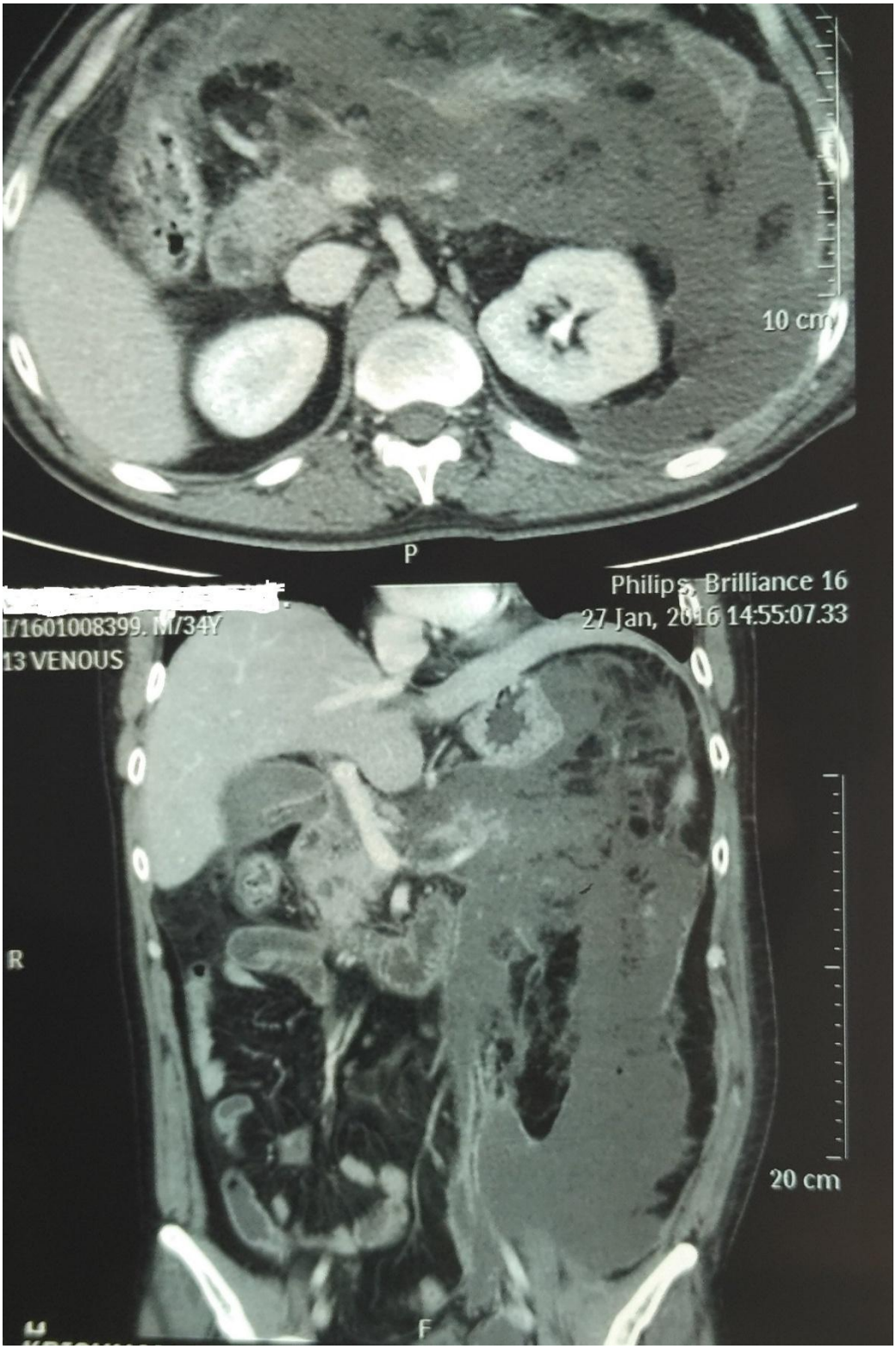


Fig.5. EXTENSIVE NECROSIS WITH MODS

Fig.6.NECROSECTOMY GROUP

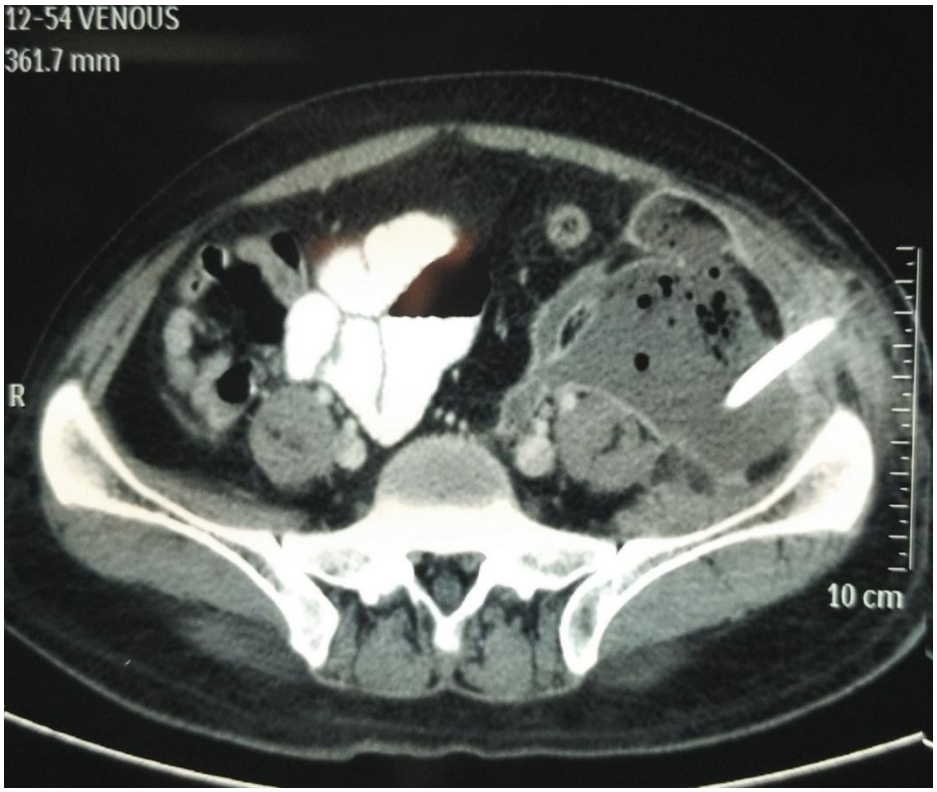
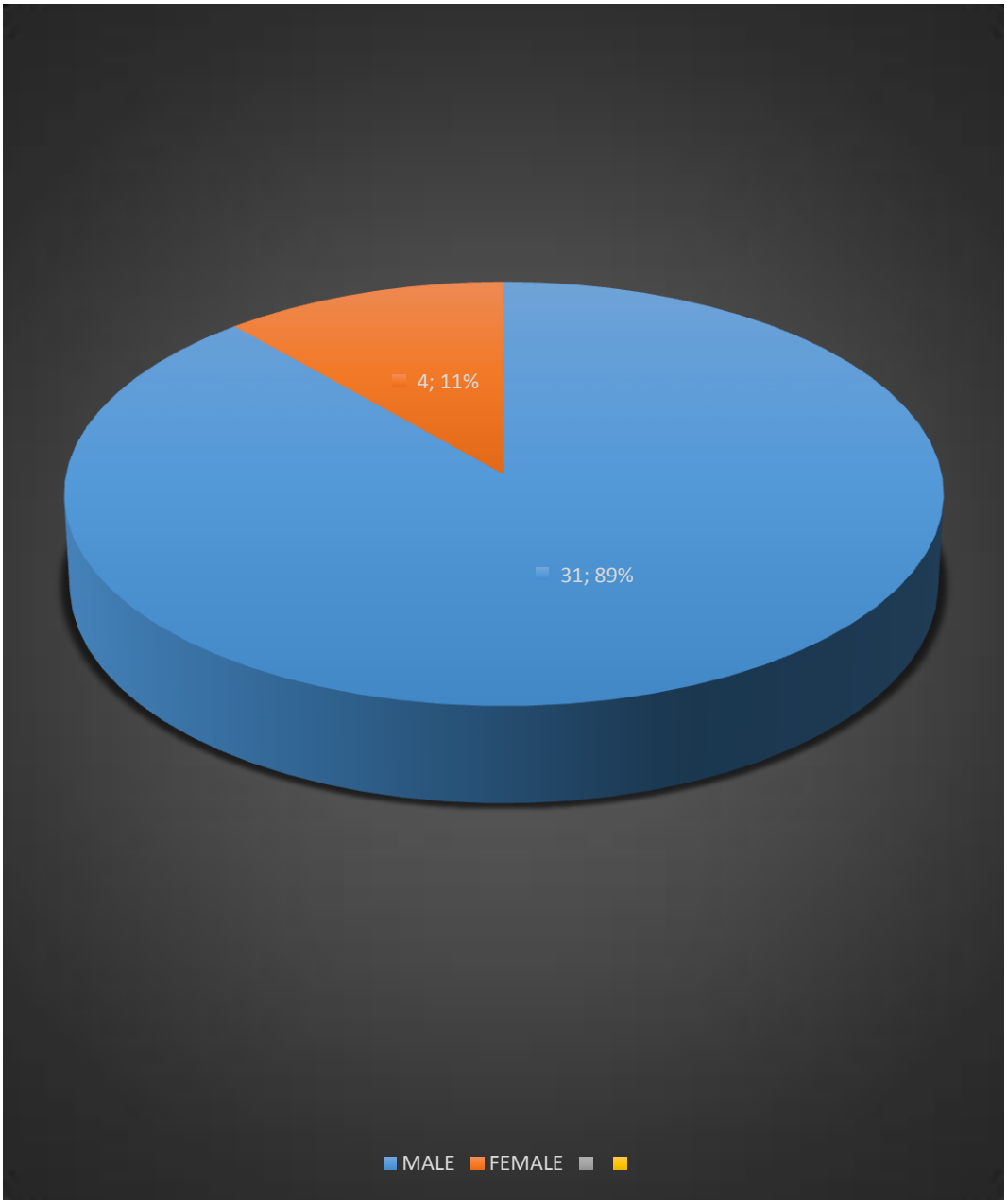
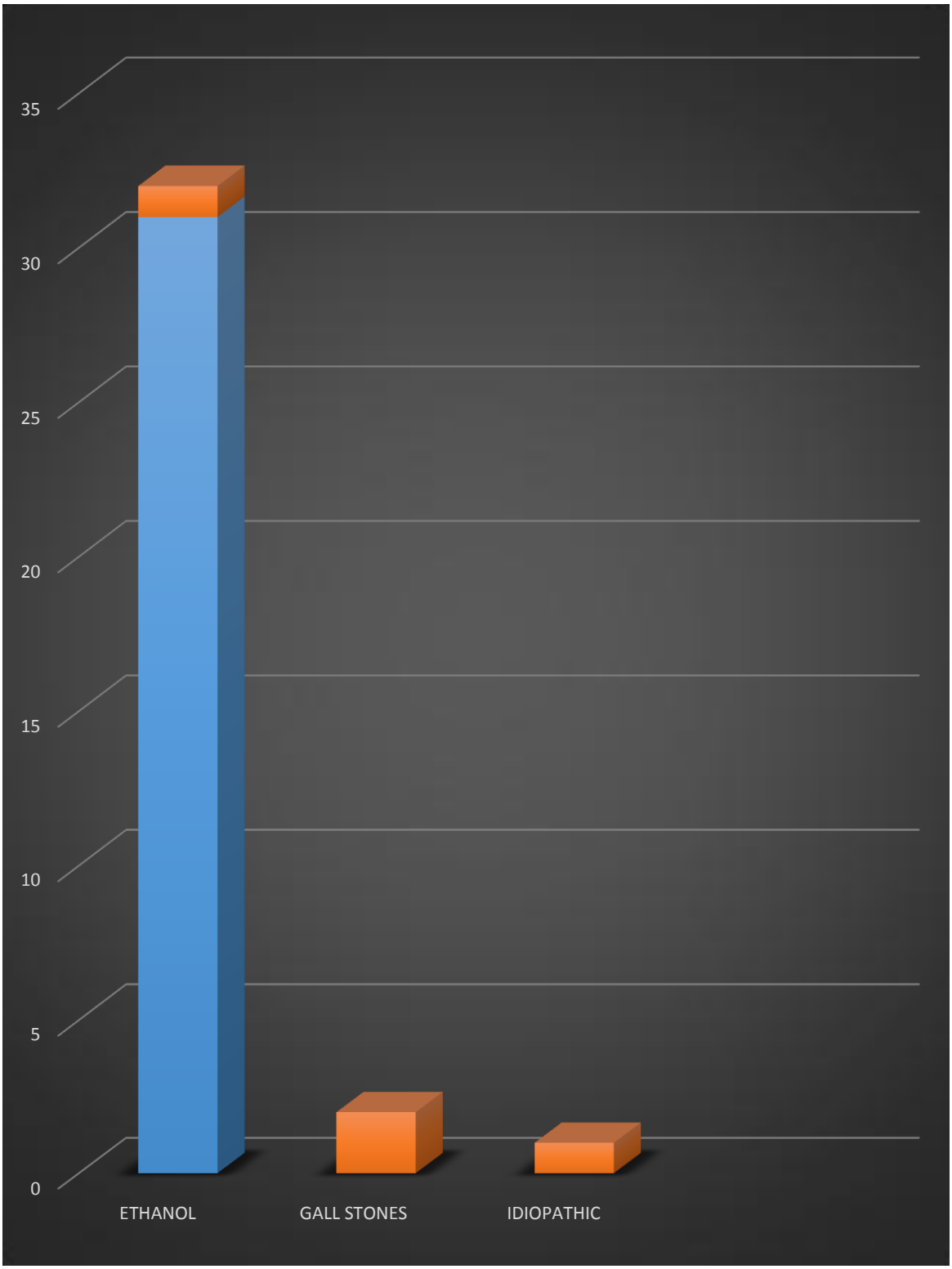


Fig.7. PCD ACTED AS A BRIDGE TO NECROSECTOMY

SEX DISTRIBUTION

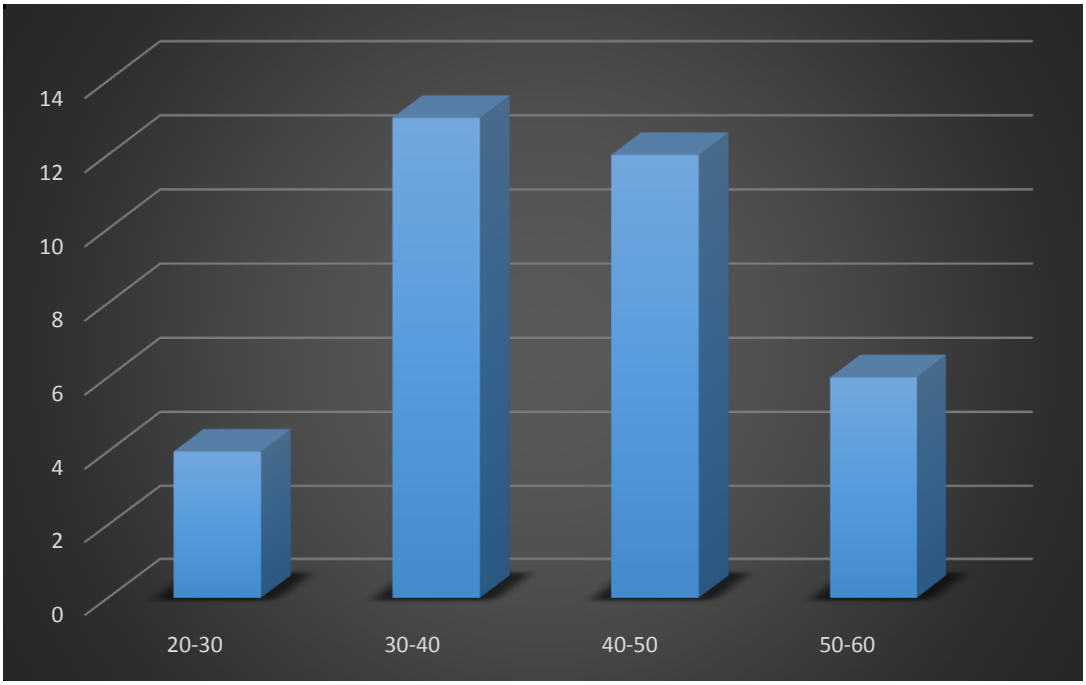


ETIOLOGY

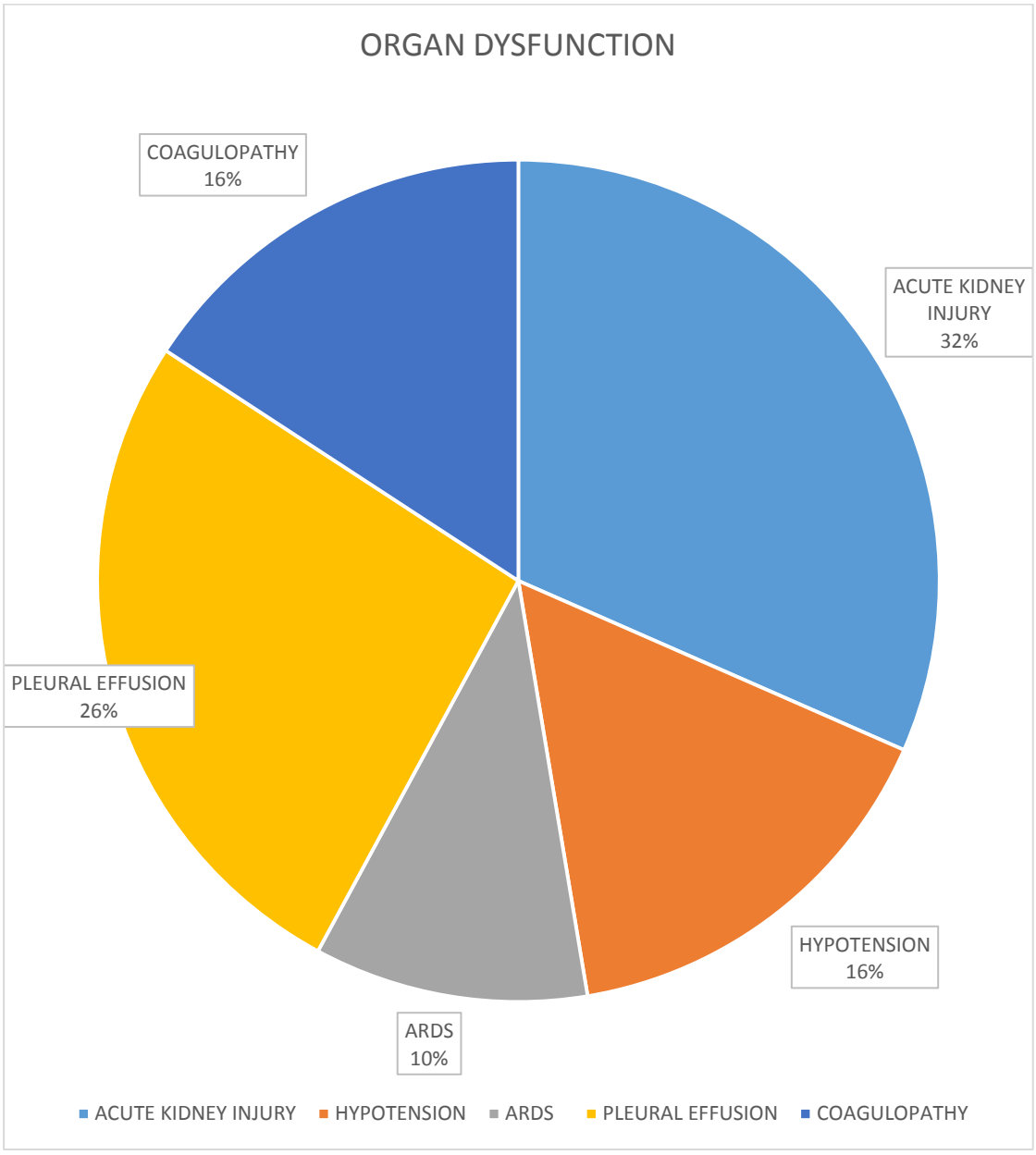


AGE DISTRIBUTION

C.I	f	x	fx	$x^{/}$	$x-x^{/}$	$(x-x^{/})^2$	$f(x-x^{/})^2$
21-30	4	25	100	40	-15	225	900
31-40	13	35	455	40	-5	25	325
41-50	12	45	540	40	5	25	300
51-60	6	55	330	40	15	225	1350
	35		**1425**				**2875**

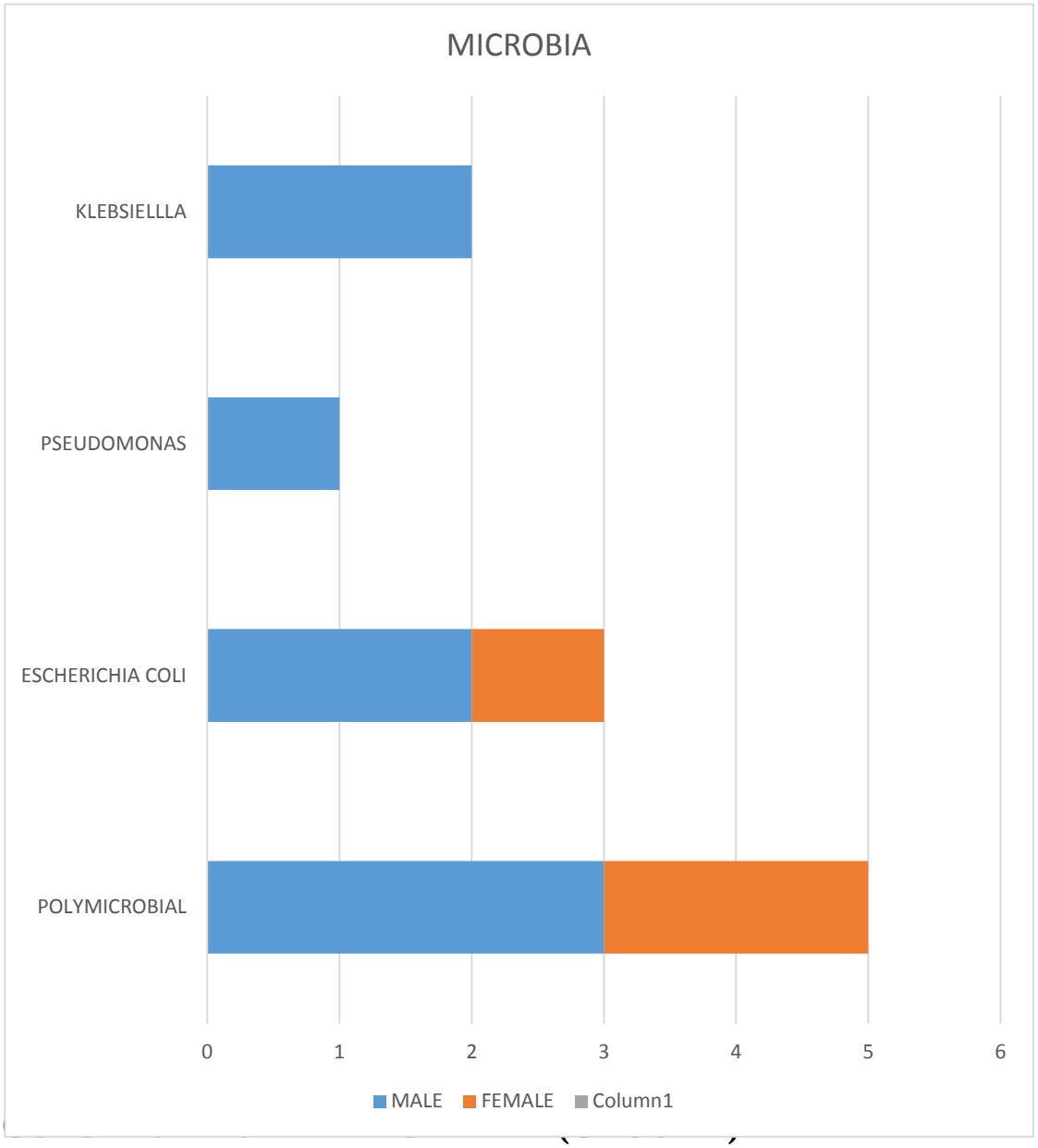


ORGAN DYSFUNCTION

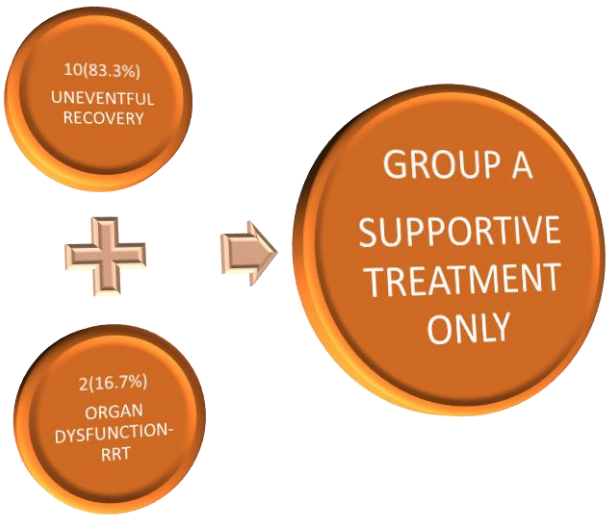


FLORA IDENTIFIED

Fine needle aspiration and gram staining was performed in those with no direct evidence of infection (absence of air inside the necrotic collections) and the following results were obtained.

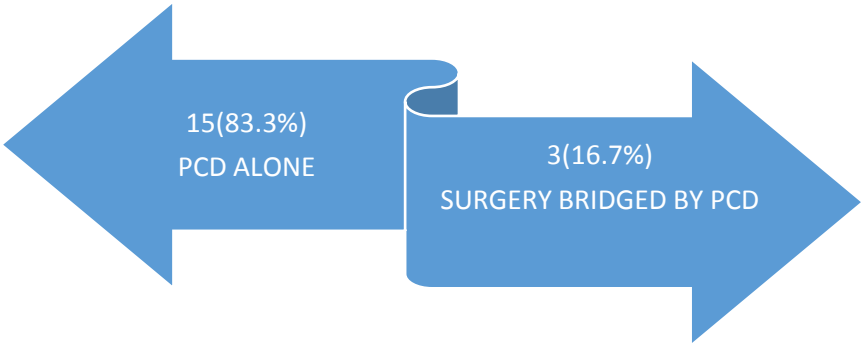


- Non-operative strategy of supportive treatment including fluid resuscitation, organ system support (including renal replacement therapy), pain alleviation and prophylactic antibiotics (imipenem/cilastatin or meropenem and metronidazole) was first used in 30 (85.7%) patients.
- 12(340.3%) cases were successfully treated by conservative management only (Group A) and 18(51.4) cases were treated with conservative approach and PCD (Group B).
- 2(16.7%) patients in Group A had organ dysfunction complication in the form of acute kidney injury and acute lung injury. 10(83.3%) patients in Group A made an uneventful recovery. 2(16.7%) patients recovered after renal replacement therapy in the form of hemodialysis



PERCUTANEOUS CATHETER DRAINAGE (GROUP B)

- 18(51.4%) patients underwent ultrasound or CT-guided drainage of the peripancreatic collections. 15(83.3%) of these patients were successfully managed by PCD drainage alone, whereas 3(16.7%) patients did not show consistent improvement with PCD alone and hence underwent laparotomy and necrosectomy thereby migrating to Group B2.
- The most common procedure related complications were catheter slippage which required repositioning in 2(11.1%) patients.



NECROSECTOMY AND CLOSED LESSER SAC LAVAGE (GROUP C)

- 5(14.3%) patients underwent surgical management in the form of necrosectomy and closed lesser sac lavage due to a inadequacy of conservative management (Group C) and 2(11.1%) image guided PCD (Group B2).
- The main indications for surgical management included infected necrotizing pancreatitis in 3(60%) patients, multi-organ dysfunction in 1(20%) patient and Warshaw's theory of un-wellness in 1(20%) patient.
- Progressive multi-organ dysfunction was the main indication for the 2(11.1%) patients who underwent surgery after inadequate percutaneous catheter drainage (Group B2).
- There wasn't any significant difference between the individuals who underwent surgical management primarily or after inadequate percutaneous catheter drainage end organ damage (acute kidney injury) (P=1.000), acute lung injury (P=0.715) and duration of in-patient management (P=0.583).
- Patients in Group B2 had delayed surgical intervention when compared to the patients in Group C, however this difference was not statistically significant (P=0.133).

- The CT severity index among the patients who were first managed conservatively (Groups A and B1) and those patients who underwent surgical management (Group B2 and Group C) was similar and not statistically significant (P=0.185).
- Instance of acute kidney injury during in-patient stay was significantly greater (P=0.008) in the individuals treated by surgical management in comparison to those who underwent conservative management.
- The intensive care management in patients managed non-operatively and those who underwent surgery was in the range of 4-25 days and 9-35 days and the mean ± standard deviation was 6.4±7.8 days and 13.7±16.3 days.
- The hospital stay in non-operated patients and those who underwent surgery was in the range of 16-38 days and 17-69 days and the mean ± standard deviation was 26±5.4 days and 43.2±11.7 days.
- Individuals treated conservatively had a significantly decreased SICU period (P=0.002) and subsequently a decreased in-patient period (P=0.003).

- The overall case fatality rate was in 3(8.6%) patients with 2(5.7%) deaths occurring after surgery and 1(2.9%) patient died in the Group B due an acute coronary event.
- Amongst those patients managed by supportive management alone there was no mortality. Amongst the operated patients, both the deaths occurred in patients primarily operated upon (Group C) and none from inadequate percutaneous catheter drainage group (Group B2).

DISCUSSION

- Percutaneous catheter drainage not only decreases the necessity of surgical management but also reduces the disease severity and the development and progression of organ failure.
- This study shows that PCD was used successfully in 18 (51.4%) of the total 35 cases. Of the 18 patients, 15(42.9) patients recovered without the need for surgical management and it helped in delaying the morbidity of upfront surgery in 2 (11.1%) patients.
- Freeny[32] and colleagues stated that 47% of their cases were successfully treated by 'PCD alone'. Moreover less than 25% of their cases needed management by surgery.
- Navalho[33] and colleagues reported that percutaneous catheter drainage cured 63% of their patients with 33% of their patients necessitating necrosectomy after PCD failed to show any clinical, biological and radiological improvement.
- Moertle *et al*.[34] revealed that PCD procedure has the potential to be the bridging gap to surgical intervention in seven of their thirteen cases with contaminated pancreatic necrosis.
- This study strengthens their observations. PCD treatment therefore can help in stabilizing the individuals for a critical period that

could help in postponing a morbid upfront surgery or altogether avoiding surgical intervention.

- However, a study by Rocha et al.[36] stated that the use of image guided intervention will not benefit patients suffering from necrotizing pancreatitis with concomitant organ failure.
- Bruennler *et al.*[35] reported that PCD done using multiple larger bore drainage catheters has the advantage that these drains would help perform a guided percutaneous or fistulous tract necrosectomy although most of them required subsequent open necrosectomy and therefore concluded that large bore drainage has no advantage in controlling the septic focus.
- Moertele[32] and colleagues used an average pigtail catheter of 12 Fr size with no mortalities. This study uses a 12Fr. Pigtail catheter for PCD and is in accordance with the above studies.
- The complications of PCD include introduction of infection, bleeding, perforation of the hollow viscus or other vital structures and slippage of the catheter.
- Bleeding is a rare event except in patients with coagulopathy (as indicated by an elevated PT/INR) and is usually due to the disease process itself rather than PCD therapy. Arterial pseudo-aneurysms is treated by arterial embolization whereas venous bleeding is rarely catastrophic.

- ❖ Fistulization of the hollow viscus could be due to an inadvertent bowel injury during catheter insertion but is more commonly due to the spread of peri-pancreatic inflammation.
- ❖ Catheter slippage occurred in 2(11.1%) of our patients and image guided repositioning was all that was required to tackle this complication.
- ❖ A new entrant in the treatment algorithm of SAP is the use of upper gastrointestinal endoscopy to drain infected peri-pancreatic collections. A recent trial demonstrated a success rate of 80% with trans-gastric upper GI endoscopic necrosectomy and a complications rate of 26%.
- ❖ Upper GI endoscopy techniques and PCD offer a safe and an effective treatment, being a valuable addition to the armamentarium of severe acute pancreatitis management.
- ❖ In conclusion, this study reiterates the value of PCD in the management of patients with SAP where majority of our cases were managed successfully by non-operative strategies, thus obviating a morbid upfront surgical management.
- ❖ PCD bridges a critical morbid period and buys an invaluable time frame which help the patient stabilize before progression to surgical necrosectomy.

- Surgical measures are definitely essential in those where the disease process could not be tackled by a supportive and percutaneous catheter drainage strategy.
- Thus, a multimodality approach involving conservative treatment, percutaneous catheter drainage and timely surgical necrosectomy is quintessential in the treatment of individuals with severe acute pancreatitis.

CONCLUSIONS

The initial treatment of acute severe pancreatitis has changed from an initial surgical management to a more conservative approach of supportive care and minimally invasive therapy. This paradigm shift in SAP treatment is a result of path-breaking trials made by various investigators who concluded that a high case fatality rate occur after early surgical management. Amongst all complications, infected pancreatic necrosis (IPN) is, by far, the most dreaded and the most severe, accounting for a major cause of mortality associated with acute pancreatitis. The risk of contamination by bacteria increases with the duration of the disease, reaching a peak during the third week with an incidence rate of 71%. It is generally accepted that, in IPN, the contaminated non-vital 'fluid under pressure' has to be debrided to control septicemia. Also the sterile fluid collections causing symptoms such as abdominal compartment syndrome, compression symptoms such as jaundice, duodenal obstruction need to be addressed

Modern treatment algorithms of SAP involves the so-called ''step-up'' approach where percutaneous catheter drainage serves as a primary stabilizing measure and is often regarded as a temporizing method to tackle septicemia and prolong surgery free interval. The next step is the minimal access necrosectomy or the traditional open necrosectomy. That

said, in well selected patients, PCD with appropriate caliber drains and supplementary therapy is all that is required or it acts as a bridge to a delayed necrosectomy which significantly has a low morbidity and mortality when compared to an early surgery

BIBLIOGRAPHY

1. Shaheen NJ, Hansen RA, Morgan DR, et al: The burden of gastrointestinal and liver diseases, 2006. *Am J Gastroenterol* 101:2128, 2006.

2. Banks PA, Freeman ML: Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 101:2379, 2006.

3. Besselink MG, Van Santvoort HC, Boermeester MA, et al: Timing and impact of infections in acute pancreatitis. *Br J Surg* 96:267,2009.

4. Petrov MS, Shanbhag S, Chakraborty M, et al: Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. *Gastroenterology* 139:813, 2010.

5. Mao EQ, Tang YQ, Fei J, et al: Fluid therapy for severe acute pancreatitis in acute response stage. *Chin Med J (Engl)* 122:169,2009.

6. Van Santvoort HC, Besselink MG, Bakker OJ, et al: A step-up approach or open necrosectomy for necrotizing pancreatitis. *N Engl J Med* 362:1491, 2010

7. Eckerwall GE, Tingstedt BB, Bergenzaun PE, et al: Immediate oral feeding in patients with mild acute pancreatitis is safe and may accelerate recovery: A randomized clinical study. *Clin Nutr* 26:758,2007.

8. Petrov MS, Zagainov VE. Influence of enteral versus parenteral nutrition on blood glucose control in acute pancreatitis: A systematic review. *Clin Nutr* 26:514, 2007.

9. Petrov MS, Loveday BP, Pylypchuk RD, et al: Systematic review and meta-analysis of enteral nutrition formulations in acute pancreatitis. *Br J Surg* 96:1243, 2009.

10. De Vries AC, Besselink MGH, Van der Kraats CIB, et al: Antibiotic prophylaxis in acute necrotizing pancreatitis: Methodological quality of randomised controlled trials in relation to outcome. *Gut* 54:A38, 2005.

11. Wittau M, Mayer B, Scheele J, et al: Systematic review and metaanalysis of antibiotic prophylaxis in severe acute pancreatitis. *Scand J Gastroenterol* 46:261, 2011.

12. Luiten EJ, Hop WC, Lange JF, et al: Controlled clinical trial of selective decontamination for the treatment of severe acute pancreatitis. *Ann Surg* 222:57, 1995.

13. Van Santvoort HC, Besselink MG, Timmerman HM, et al: Probiotics in surgery. *Surgery* 143:1, 2008.

14. Mier J, Luque-de León E, Castillo A, et al: Early versus late necrosectomy in severe necrotizing pancreatitis. *Am J Surg* 173:71, 1997.

15. Cheatham ML, Malbrain ML, Kirkpatrick A, et al: Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med* 33:951, 2007.

16. Besselink MG, Van Santvoort HC, Bakker OJ, et al: Draining sterile fluid collections in acute pancreatitis? Primum non nocere! *Surg Endosc* 25:331, 2011.

17. Zerem E, Imamovic G, Omerovic S, et al: Randomized controlled trial on sterile fluid collections management in acute pancreatitis: Should they be removed? *Surg Endosc* 2009 May 15.

18. Petrov MS, Van Santvoort HC, Besselink MG, et al: Early endoscopic retrograde cholangiopancreatography versus conservative management in acute biliary pancreatitis without cholangitis: A meta-analysis of randomized trials. Ann Surg 247:250, 2008.

19. Van Santvoort HC, Besselink MG, De Vries AC, et al: Early endoscopic retrograde cholangiopancreatography in predicted severe acute biliary pancreatitis: A prospective multicenter study. Ann Surg 250:68, 2009.

20. Besselink MG, Verwer TJ, Schoenmaeckers EJ, et al: Timing of surgical intervention in necrotizing pancreatitis. Arch Surg 142:1194,2007.

21. Van Baal MC, Van Santvoort HC, Bollen TL, et al: Systematic review of percutaneous catheter drainage as primary treatment for necrotizing pancreatitis. Br J Surg 98:18, 2011.

21. Horvath K, Freeny P, Escallon J, et al: Safety and efficacy of video assisted retroperitoneal debridement for infected pancreatic collections: A multicenter, prospective, single-arm phase 2 study.Arch Surg 145:817, 2010.

22. Horvath KD, Kao LS, Wherry KL, et al: A technique for laparoscopic assisted percutaneous drainage of infected pancreatic necrosis and pancreatic abscess. Surg Endosc 15:1221, 2001.

23. Van Santvoort HC, Besselink MGH, Horvath KD, et al: Videoscopic assisted retroperitoneal debridement in infected necrotizing pancreatitis. HPB (Oxford) 9:156, 2007.

24. Raraty MG, Halloran CM, Dodd S, et al: Minimal access retroperitoneal pancreatic necrosectomy: Improvement in morbidity and mortality with a less invasive approach. Ann Surg 251:787,2010.

25. Papachristou GI, Takahashi N, Chahal P, et al: Peroral endoscopic drainage/debridement of walled-off pancreatic necrosis. Ann Surg 245:943, 2007.

26. Seifert H, Biermer M, Schmitt W, et al: Transluminal endoscopic necrosectomy after acute pancreatitis: A multicentre study with long-term follow-up (the GEPARD Study). Gut 58:1260, 2009.

27. Voermans RP, Bruno MJ, van Berge Henegouwen MI, et al: Review article: Transluminal endoscopic debridement of organized pancreatic necrosis—the first step towards natural orifice transluminal endoscopic surgery. Aliment Pharmacol Ther 26:233, 2007.

28. Besselink MG, De Bruijn MT, Rutten JP, et al: Surgical intervention in patients with necrotizing pancreatitis. Br J Surg 93:593,2006.

29. Rau B, Bothe A, Beger HG. Surgical treatment of necrotizing pancreatitis by necrosectomy and closed lavage: Changing patient characteristics and outcome in a 19-year, single-center series. Surgery 138:28, 2005**.**

30. Rodriguez JR, Razo AO, Targarona J, et al: Debridement and closed packing for sterile or infected necrotizing pancreatitis: Insights into indications and outcomes in 167 patients. Ann Surg 247:294, 2008.

31. Nealon WH, Bawduniak J, Walser EM. Appropriate timing of cholecystectomy in patients who present with moderate to severe gallstone-associated acute pancreatitis with peripancreatic fluid collections. Ann Surg 239:741, 200

32. Freeny PC, Hauptmann E, Althaus SJ, Traverso LW, Sinanan M. Percutaneous CT-guided catheter drainage of infected necrotizing pancreatitis: techniques and results. AJR Am J Roentgenol 1998; 170:969-75. [PMID 9530046]

33. Navalho M, Pires F, Duarte A, Gonçalves A, Alexandr ino P, Távora I. Percutaneous drainage of infected pancreatic fluid collections in critically ill patients: correlation with C-reactive protein values. Clin Imaging 2006; 30:114-9. [PMID 16500542]

34. Mortelé KJ, Girshman J, Szejnfeld D, Ashley SW, Erturk SM, Banks PA, Silverman SG. CT-guided percutaneous catheter drainage of acute necrotizing pancreatitis: clinical experience and observations in patients with sterile and infected necrosis. AJR Am J Roentgenol 2009; 192:110-6. (PMID 19098188)

35. Bruennler T, Langgartner J, Lang S, Wrede CE, Klebl F, Zierhut S, et al. Outcome of patients with acute necrotizing pancreatitis requiring drainage - does drainage size matter. World J Gastroenterol 2008; 14:725-30. (PMID 18205262)

36. Rocha FG, Benoit E, Zinner MJ, Whang EE, Banks PA, Ashley SW, Mortele KJ. Impact of radiologic intervention on mortality in necrotizing pancreatitis: the role of organ failure. Arch Surg 2009; 144:261-5. [PMID 19289666]

37. Warshaw AL. Pancreatic necrosis: to debride or not to debride-that is the question. Ann Surg. 2000 Nov;232(5):627–629.

PROFORMA

PATIENT DETAILS

Age: Sex: IP No. :

ON ADMISSION

Presenting complaints :

Past history:

Personal history:

Treatment history:

CLINICAL EXAMINATION

Pulse : BP :

RR: Temperature:

Pallor: Icterus:

CVS: RS:

P/A: CNS:

INVESTIGATIONS

CBC/RFT				
TC,DC				
PT/INR				
Hb %				
PCV				
RBC				
Platelets				
Glucose				
Urea				
Creatinine				
Na^+				
K^+				
LFT				
Total Bilirubin				
Dir. Bilirubin				
SGOT				
SGPT				
SAP				
Total Protein				
Sr. Albumin				
Sr.amylase				
Sr.lipase				
CRP				

Chest Xray:

Abdomen Xray:

USG Abdomen:

CECT Abdomen:

Provisional diagnosis with etiology:

TREATMENT GROUP

Conservative Management (Group A)

Percutaneous Catheter Drainage (Group B)

Surgical Management (Group C)

Intra Op findings: Post op Period :

FOLLOW UP

S.NO.	IP.NO.	AGE	SEX	CAUSE OF SAP	AKI	ARDS	PLEURAL EFFUSION	COAGULOPATHY	HYPOTENSION	CTSI	GROUP	MODE OF PCD	NATURE OF COLLECTION	FLORA IDENTIFIED	NO. OF DAYS OF DRAINAGE	NO.OF DAYS OF HOSTITAL STAY
1	14951	25	M	A	Y	Y	Y	N	N	8	B,C	USG	Inf.	Poly	5	48
2	15423	21	M	A	Y	N	N	N	Y	8	C	-	-	-	-	54
3	15689	38	F	I	N	N	N	N	N	7	B	USG	Ster.	-	6	29
4	12218	30	M	A	N	N	N	N	N	7	A	-	-	-	-	16
5	26459	38	M	A	N	N	N	N	N	8	B	USG	Inf.	E.coli	8	25
6	26558	49	M	A	N	N	N	N	N	7	A	-	-	-	-	14
7	36789	41	M	A	N	N	N	N	N	8	B	USG	Inf.	Poly	1	26
8	42158	39	M	A	N	N	N	N	N	7	A	-	-	-	-	18
9	46987	47	M	A	N	N	N	N	N	7	B	USG	Inf.	E.coli	8	25
10	49784	37	M	A	Y	N	N	N	N	8	C	-	-	-	-	17
11	54158	45	F	A	N	N	N	N	N	7	B	USG	Ster.	-	-	24
12	64789	52	M	A	N	N	N	N	N	6	A	-	-	-	-	16
13	68794	52	M	A	N	N	Y	Y	Y	9	B,C	CT	Inf.	Pseud	6	50
14	74158	24	M	A	N	N	N	N	N	6	B	USG	Ster.	-	-	21
15	79145	50	M	A	N	N	N	N	N	6	A	-	-	-	-	19
16	80698	32	M	A	N	N	N	N	N	7	B	USG	Ster.	-	-	21
17	81074	35	M	A	N	N	N	N	N	6	A	-	-	-	-	12
18	81186	43	M	A	N	N	N	N	N	7	B	USG	Inf.	Poly	10	25
19	81204	48	M	A	Y	N	N	N	N	7	B	USG	Ster.	-	-	23
20	85478	42	M	A	N	N	N	N	N	6	A	-	-	-	-	11

S.NO.	IP.NO.	AGE	SEX	CAUSE OF SAP	AKI	ARDS	PLEURAL EFFUSION	COAGULOPATHY	HYPOTENSION	CTSI	GROUP	MODE OF PCD	NATURE OF COLLECTION	FLORA IDENTIFIED	NO. OF DAYS OF DRAINAGE	NO.OF DAYS OF HOSTITAL STAY
21	89745	41	M	A	N	N	N	N	N	6	A	-	-	-	-	10
22	91245	54	M	A	N	N	N	N	N	8	B	USG	Inf.	E.coli	9	26
23	93214	48	M	A	Y	N	Y	Y	N	9	C	-	-	-	-	46
24	94152	40	M	A	N	N	N	N	N	7	B	USG	Inf.	Kleb.	6	23
25	96451	28	F	G	N	N	N	N	N	6	A	-	-	-	-	10
26	97451	36	M	A	N	N	N	N	N	7	B	USG	Ster.	-	-	19
27	99748	45	M	A	N	N	N	N	N	6	A	-	-	-	-	13
28	100111	35	M	A	N	N	N	N	N	8	B	USG	Inf.	Kleb.	8	25
29	111246	40	M	A	N	N	Y	N	N	10	C	-	-	-	-	40
30	132148	55	M	A	N	N	N	N	N	6	A	-	-	-	-	11
31	146875	60	M	A	N	N	N	N	N	10	C	-	-	-	-	39
32	164474	33	M	A	N	N	N	N	N	8	B	USG	Inf.	Poly	8	23
33	187459	34	M	A	N	N	N	N	N	6	A	-	-	-	-	13
34	211541	50	M	A	N	N	N	N	N	7	B	USG	Ster.	-	-	27
35	252145	60	F	G	Y	Y	Y	Y	Y	10	B,C	CT	Inf.	Poly	4	69

LEGEND

M/F-MALE/FEMALE

Y/N-YES/NO

A-ALCOHOL

G-GALL STONE

I-IDIOPATHIC

INF-INFECTED COLLECTION

STER-STERILE COLLECTION

E.coli-ESCHERICHIA COLI

PSEU-PSEUDOMONAS sp.

KLEB-KLEBSIELLA sp.

POLY-POLYMICROBIAL

INFORMATION SHEET

TITLE: ***"A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS"***

Name of Investigator: Dr. J.MOHAMMED FAROOQ. **Name of Participant**:

Purpose of Research: To study the role of percutaneous catheter drainage in the management of acute necrotizing pancreatitis and to determine the factors influencing its clinical success

Study Design: Prospective Observational Study

Study Procedures: Patient will be subjected to routine investigations, Xray, Usg, CECT Abdomen, complete hemogram, percutaneous catheter drainage procedure as indicated, and the data analysed

Possible Risks: No risks to the patient

Possible benefits

To patient : A better understanding of their problem so has to devise a plan of management which suits their needs.

To doctor & to other people: If this study gives positive results, it can help determine the role of percutaneous catheter drainage (minimally invasive procedure) in the treatment of patients with severe pancreatitis. This will help in providing better and complete treatment to other patients in future.

Confidentiality of the information obtained from you: The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared

Can you decide to stop participating in the study: Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you: Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail : ***"A STUDY OF THE ROLE OF PERCUTANEOUS CATHETER DRAINAGE IN THE MANAGEMENT OF ACUTE SEVERE PANCREATITIS"***

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

In Patient Number :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment ❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

Dr. J, MOHAMMED FAROOQ

<u>ஆய்வு ஒப்புதல் படிவம்</u>

ஆய்வின் தலைப்பு

கடுமையான கணைய அழற்சி நோயிற்கு தோல் மூலமாக வடிகால் சிகிச்சையின் பயனுடைமை பற்றி அறிவதற்கான ஆய்வு

ஆய்வு நிலையம் : பொது அறுவை சிகிச்சைத்துறை, ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை மருத்துவக் கல்லூரி சென்னை – 3.

பங்கு பெறுவரின் பெயர் :

பங்குபெறுபவரின் எண் :

பங்குபெறுபவர் இதனை (✓) குறிக்கவும்

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக அறிந்துகொண்டேன். எனது சந்தேகங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுய நினைவுடன் பங்குகொள்ள சம்மதிக்கிறேன். [ ]

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது. [ ]

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்துகொண்டேன். [ ]

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன். [ ]

இந்த ஆய்வில் கலந்துகொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்துகொள்ளலாம் என சம்மதிக்கிறேன். [ ]

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரையோ, அடையாளங்களையோ வெளியிடப்படாது என அறிந்துகொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக்கொண்டேன். இந்த ஆய்விற்காக இரத்தப் பரிசோதனை செய்துகொள்ள சம்மதிக்கிறேன். [ ]

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்புகொள்ள வேண்டும் என அறிந்துகொண்டேன். [ ]

இந்த ஆய்வில் எனக்கு மருத்துவ பரிசோதனை, இரத்தப் பரிசோதனை மற்றும் இதய உட்பு ஆய்வு பரிசோதனை செய்துகொள்ள முழு மனதுடன் சம்மதிக்கிறேன். [ ]

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்று தெரிவிக்கிறேன் என்று புரிந்துகொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்று தெரிந்துகொண்டேன். [ ]

பங்கேற்பாளர்/ பாதுகாவலர் கையொப்பம் தேதி:

ஆய்வாளர் கையொப்பம் தேதி:

ஆய்வில் பங்கேற்பவருக்கான தகவல் அறிக்கை

ஆய்வு தலைப்பு

கடுமையான கணைய அழற்சி நோயிற்கு தோல் மூலமாக வடிகால் சிகிச்சையின் பயனுடைமை பற்றி அறிவதற்கான ஆய்வு

பங்குகொள்பவரின் பெயர் :

ஆய்வு செய்பவரின் பெயர் : மரு.ஜே.முகமது பாருக்

இடம் : ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை-600 003.

இந்த ஆய்வின் நோக்கம் என்ன?

நம் நாட்டில் கணைய அழற்சி நோய் அதிகமாக உள்ளது. அதற்கு பல்வேறு காரணங்கள் உள்ளன. இந்த ஆய்வில் ராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் கணைய அழற்சி நோயிற்கு பல்வேறு பரிசோதனைகள் மற்றும் சிகிச்சைகள் அளிக்கப்பட்டு வருகின்றன. இவற்றில் கடுமையான கணைய அழற்சி நோயிற்கு தோல் மூலமாக வடிகால் சிகிச்சையின் பயனுடைமையை பற்றி அறிவதே இந்த ஆய்வின் நோக்கமாகும். மேலும் கணைய அழற்சியினால் ஏற்படும் நோய் நுண்மங்கள் நிறைந்த மற்றும் நோய் நுண்மங்கள் அற்ற திரட்டுகளுக்கு தோல் மூலமாக வடிகால் சிகிச்சையின் முடிவுகளை ஒப்பிடும் ஆராய்ச்சி.

ஆய்வு முறைகள்

விரிவான நோய் குறிப்புகளும், மருத்துவ பரிசோதனை மற்றும் சிகிச்சை நோயாளிகள் அவர்கள் சம்மதத்திற்கு இணங்க செய்யப்பட்டு அதன் பயனுடைமைகள் மற்றும் பலாபலன்கள் ஆராயப்படும்.

ஆய்வினால் மக்களுக்கு ஏற்படும் நன்மைகள்

இந்த ஆய்வின் முடிவில் கிடைக்கும் தகவல்கள் சமுதாயத்திற்கு பயனுள்ளதாகவும், எதிர்காலத்தில் நோயாளிகளுக்கு மருத்துவ தீர்வாகவும் அமையும்.

தங்களிடமிருந்து பெறப்படும் தகவல்களின் நம்பகத்தன்மை

தங்களிடமிருந்து பெறப்படும் தகவல்கள் பாதுகாக்கப்படுவதற்கான முழு உரிமையும் தங்களுக்கு உண்டு.

இந்த படிவத்தில் கையொப்பமிடுவதின் மூலம், தாங்கள் தங்களைப் பற்றிய விவரங்களையும், ஆய்வு விவரங்களையும் ஆராய்ச்சியாளர், ஆய்வு நடத்தும் ஏனையோர் வரைமுறை ஒழுங்கு குழுவினர் மற்றும் சட்டத்திற்கு உட்பட்டு மருந்து கட்டுப்பாடு இயக்குநர் ஆகியோர் பார்வையிட அனுமதிக்கிறீர்கள்.

இந்த ஆய்வில் காட்டப்படும் தகவல்கள் அறிவியல் நாளேடுகளிலோ அறிவியல் கூட்டங்களிலோ சமர்ப்பிக்கப்படும் பட்சத்தில் தங்களது அடையாளம் வெளிப்படுத்தப்படமாட்டாது.

இந்த ஆய்வில் பங்கேற்காமல் இருப்பதினால் ஏற்படும் பாதிப்பு

இந்த ஆய்வில் தாங்கள் பங்கேற்க விருப்பம் தெரிவிக்காத நிலையில் தங்களின் மருத்துவர் மற்றும் மருத்துவமனையில் தங்களுக்கு உள்ள உறவில் எந்த பாதிப்பும் ஏற்படாது. தாங்கள் சிறப்பாக கவனிக்கப்படுவீர்கள். மேலும் இதனால் தங்களுக்கு இழப்பு ஏதும் ஏற்படாது.

ஆய்வின் நடுவில் அதிலிருந்து விலகிக்கொள்ள நினைத்தால்

இந்த ஆய்வில் பங்கேற்பது தங்களின் சொந்த விருப்பமே. மேலும் ஆய்வின் நடுவில் எந்த நேரத்திலும், எக்காரணமும் கூறாமல் விலகிக்கொள்ள தங்களுக்கு முழு உரிமையும் உண்டு. இருப்பினும் ஆய்விலிருந்து விலகுவதற்கு முன் ஆராய்ச்சி குழுவுடன் கலந்து ஆலோசிப்பது உகந்தது என பரிந்துரைக்கப்படுகின்றது.

ஆராய்ச்சியாளர் கையொப்பம்
தேதி:

பங்கேற்பாளர் கையொப்பம்
தேதி: